Econometric models of child mortality dynamics in rural Bangladesh

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Econometric Models of Child Mortality Dynamics in Rural Bangladesh

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Proefschrift

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> “Look at the sky. We are not alone. The whole universe is friendly to us and conspires only to give the best to those who dream and work” **A.P.J. Abdul Kalam, ex-president of India**

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Chapter 1

Introduction

Health transition is an important aspect of demographic change and a complex process comprising demographic, epidemiological and health care transitions. It is manifested in rising life expectancy at birth due to changes in the fertility, mortality and morbidity profile of a population. Demographic transition brings down birth and death rates and changes the age structure; epidemiological transition reflects changes in the causes of death, from infectious (pandemic) diseases to non-communicable (degenerative, human-made) diseases (Caldwell et al. 1990; Omran 1982). However, the causal mechanisms of demographic changes are unclear. The focus of my thesis is to uncover these causal mechanisms, and to quantify the epidemiological shifts over time, taking into account phenomena that hamper a straightforward empirical analysis, such as competing risks, observed and unobserved confounding factors, and reverse causality. This analysis may influence policy development in countries such as Bangladesh, whose ability to meet Millennium Development Goal 4 (see United Nations 2001) is currently in doubt.

1.1 Background

According to demographic transition theory, there is a strong correlation between childhood mortality and fertility. Empirical evidence has shown that a decline in childhood mortality is often a prerequisite for fertility decline (Chowdhury et al. 1976; Matthiesson and McCann 1978; Pritchett 1994; Wolpin 1997). Other studies have emphasized the reverse direction of this causation, e.g., high fertility and the close birth-spacing associated with it cause an increase in child mortality (Cleland and Sathar 1984; Curtis et al. 1993). Yet another set of studies emphasized that the analysis of the direction of causality with birth interval data is hampered by the close interrelations between child mortality and fertility (Zimmer 1979; Santow and Bracher 1984).

Family planning is also related to health transition in multifaceted ways. Through family planning practice, a couple can decide the time of birth, the time span between two births, and the (maximum) number of children they want to have. Thus, once family size declines, there is an added incentive to ensure the survival of children. In the developing world, some success is evident in reducing child death in small families (see for example, Caldwell and Caldwell 1978). Family planning practice can also avoid births at extreme age and at short intervals, which have detrimental effects on child mortality. Yet another set of arguments posit that if the society is not

overly concerned with the sexual purity of its women, it is more likely to permit girls to go to school and even to stay there when they reach puberty, which in turn will inevitably transfer more family resources towards children and ultimately towards wives (Caldwell 1993).

There is a large literature on the determinants of childhood mortality in developing countries, focusing on, for example, the fact that children of very young mothers or mothers with little or no schooling are at higher risk. Moreover, demographic data from many countries have revealed that child deaths are clustered within families. See, for example, Das Gupta (1990) for India, Gubhaju (1985) for Nepal, Guo and Rodriguez (1992) and Guo (1993) for Guatemala, Curtis et al. (1993) and Sastry (1997) for Brazil, or Madise and Diamond (1995) for Malawi. It is argued by demographers that this small fraction of deaths may be a product of infectious disease or biological differences, but probably most of the explanation lies in different levels of family care or interaction with the health system.

However, with the available explanation and the evidence of correlated sibling death, the causal relationship of child deaths and fertility, and epidemiological transition, the empirical literature is indeed limited on areas such as the causal mechanisms in the relationship between fertility and mortality, or how to accommodate the correlation with sibling death at an aggregate level or at the level of causes of death (see for example, DaVanzo et al. 2008; Yeakey et al. 2009; Bhatia 1989). These particular issues form the focus of four separate papers included in my thesis.

1.2 The Matlab Area

Matlab Thana is an administrative region in the Chandpur district of Bangladesh. Matlab is located about 55 km southeast of Dhaka, the capital city of Bangladesh. The climate is sub-tropical with three seasons: monsoon, cool-dry and hot-dry. The average annual rainfall of 2159 mm is concentrated in the monsoon season extending from June to September. Being flat and low-lying it is subject to annual flooding by many canals and rivers which cross the area. The population density is about 1,100 per km^2 residing in 142 villages. The area is a typical rural river delta area of Bangladesh. Almost 90% of the population are Muslims and the great majority of the remainder are Hindus; all of them speak Bangla. The principle economic activities are agriculture and fishing. For most dwellings, roof material is tin (95%), while in 30% tin was used for wall material. Travel within Matlab Thana and between the villages is mostly by foot or rickshaw or country boat, particularly during the monsoon season.

Since 1963, the ICDDR,B Centre for Health and Population Research, formally Cholera Research Laboratory, has been implementing a health related research programme. The health and Demographic Surveillance System (HDSS), formally Demographic Surveillance System (DSS), is one of the major components of this field programme. Since 1966, the HDSS has been maintaining the registration of births, deaths, and migration, in addition to carrying out periodical censuses in 149 villages 70 in ICDDR,B area and 79 in the comparison area (so called

government area). Due to river erosion, 7 villages disappeared from the comparison area in 1987, leaving 142 villages in the HDSS. In 2000, 3 of the 70 villages of ICDDR,B area were transferred to the comparison area. A map is given in the Annex.

1.3 Health Systems in Matlab, Bangladesh

In 1977, the International Centre for Diarrhoeal Disease Research, Bangladesh (ICDDR,B) started to provide extensive maternal-child health and family planning (MCH/FP) services, in addition to existing government health services, in half (70 villages) of the Health and Demographic Surveillance System (HDSS) area, called the ICDDR,B area. The other half (79 villages), the comparison area, continued to receive only the standard government health services. The MCH/FP project includes provision of domiciliary family planning services, simple nutrition education, tetanus toxoid immunization for pregnant women (which was modified in 1981 to include all women of reproductive age), community-based oral rehydration therapy, and measles immunization. These services were introduced incrementally phase by phase (see Annex, Table 1). In the ICDDR,B area, there are several ICDDR,B sub-centres providing treatment for minor illnesses and basic emergency obstetric care (EOC), and a permanent hospital that provides treatment for diarrhoeal diseases. In order to understand the way in which better health services shape child health, I analyze the data from the area with the better health care services in addition to the government health services (ICDDR,B area), as well as the data from an area with standard government health services only (the comparison area - a typical rural area of Bangladesh).

1.4 Methodological issues and data

The primary focus of my dissertation is to apply an econometric approach to empirical research on child mortality dynamics in Bangladesh. In econometrics, the assumptions of the underlying population model are couched in terms of correlations, conditional expectations, and conditional variances-covariances, or conditional distributions, can usually be given behavioral content. In demographic research, the application of econometrics can bring greater insight into the behavioral context than can mere quantitative measures. Several researchers, such as Bongaarts (1987) or DaVanzo et al. (2008), saw the importance of investigating and disentangling the various causal and non-causal relationships between birth-spacing, fertility decisions and childhood mortality, in countries in demographic transition such as Bangladesh.

In social sciences research, we very often fail to include potential observed covariates in the model, which might lead to an unobserved heterogeneity (unobserved variability) in the response variable. This is a common phenomenon for cross section or panel data: observations for the same unit are influenced by the same (shared) unit-specific time invariant unobserved heterogeneity. Thus, while the major concern of statistical modeling is to explain the variability in the response variable in terms of the effects of observed covariates, called ‘observed heterogeneity’, failing to be able to include all relevant covariates due to data limitations leads to

unobserved heterogeneity. This needs to be accounted for in the analyses to obtain unbiased estimates.

Causal analysis (investigating cause and effect) is an important aspect of econometrics. Utilizing this concept in demographic research, part of my thesis investigates the bi-causal relationship between fertility and mortality, and for example, how birth spacing shapes this relationship. I also use the concept of state dependence as used in the labor economics literature on unemployment to investigate for example, the clustering of deaths of siblings. Since decisions are inherently dynamic and sequential, the static models often employed to study variables such as mortality and fertility can produce misleading estimates (Wolpin 1997; Rosenzweig and Wolpin 1988). The main motivation for this thesis is to follow the modelling framework of Arulampalam and Bhalotra (2006) and Bhalotra and van Soest (2008) in order to account for the dynamics and the simultaneous nature of mortality-birth spacing and fertility sequencing decisions, and for endowments (persistent mother-specific traits), modeled as unobserved heterogeneity components within families. A recent study on investigating the causal impact of fertility timing on education also noted the importance of such dynamic models when decisions are sequential (see Stange 2011).

All four papers on which the chapters are based, aim to analyze information on individuals (child level information) and families (mother level information) over the period 1982-2005. In the second and third chapters, information on the complete pregnancy history of a mother is used, for example, live births, deaths and several indicators of socioeconomic status, are recorded for the population of about 220,000 people in the Matlab Health and Demographic Surveillance System (HDSS) area, split into ICDDR,B and comparison area. In the fourth chapter, the modelling framework used in the third chapter is extended by including the mother's contraceptive use status after each birth. The fifth chapter uses the birth history of each mother and their live births, neonatal deaths and socio economic indicators. In this chapter, data obtained on both complete and incomplete birth histories are used, because birth spacing is not the primary interest of analysis. Although, it remains important to include birth spacing in the list of explanatory variables, it is avoided because of endogeneity problems making the modeling more complicated. It remains a topic for further research.

1.5 Research issues

The research theme of my dissertation is common and has long been of interest to demographers. Apart from previous research on this issue, using several advanced econometric techniques to answer the related research questions in all chapters, I have worked jointly with co-authors and developed four separate but related papers. All chapters of my dissertation are based on papers except the introduction in the first chapter, and the overall summary and conclusion in the last chapter. In the second chapter, we investigate sibling death clustering in families and use of better health services. We use an existing modeling framework distinguishing two explanations for death clustering: (observed and unobserved) heterogeneity across families, and a causal "scarring" effect of infant mortality of one child on the survival chances of the next child.

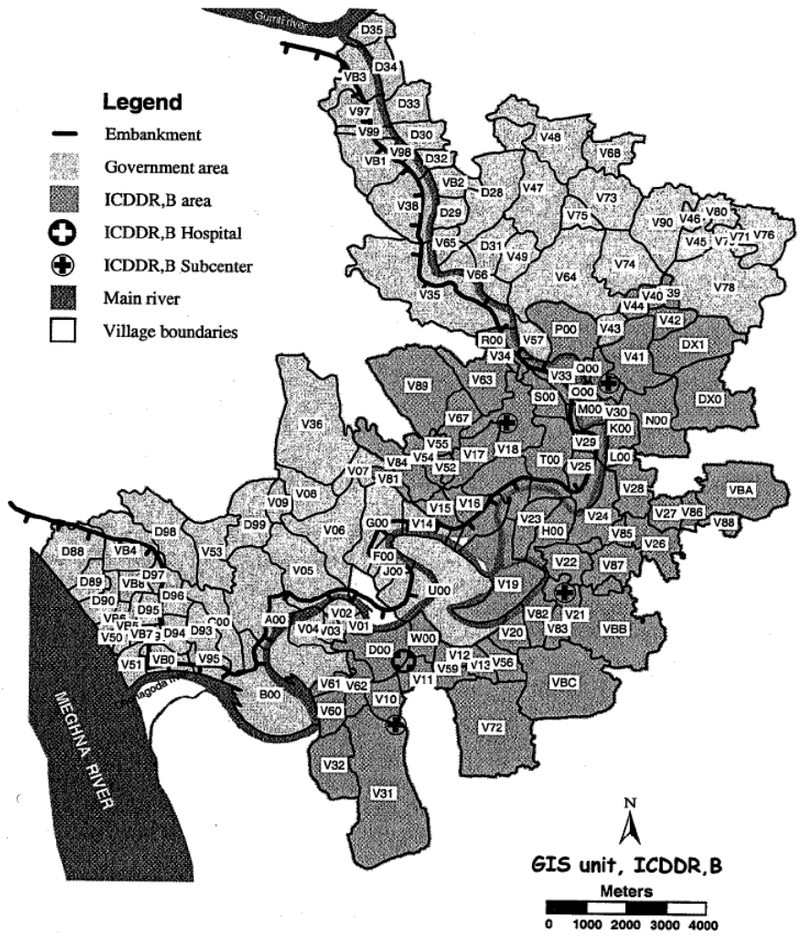
In the third chapter, to distinguish causal mechanisms from unobserved heterogeneity and reverse causality, we used dynamic panel data techniques, building on recent work by Bhalotra and van Soest (2008). This model incorporates various causal mechanisms as well as unobserved heterogeneity, exploiting the sequence of all births and deaths to a mother to accommodate the correlations between the mortality risks of consecutive children and birth intervals. We compare the results in a treatment area (the ICDDR,B area) with extensive health services and a comparison area with the standard health services provided by the government.

In the fourth chapter, we analyze the effect of family planning on child survival, which remains an important issue but is not straightforward, because of several mechanisms linking family planning, birth intervals, total fertility, and child survival. This study uses a dynamic model jointly explaining infant mortality, whether contraceptives are used after each birth, and birth intervals. Infant mortality is determined by the preceding birth interval and other covariates (such as socio-economic status). Decisions about using contraceptives after each birth are driven by similar covariates, survival status of the previous child, and the family's gender composition. Birth spacing is driven by contraceptive use and other factors.

In the fifth chapter, we focus on explaining cause-specific neonatal mortality and employ a competing risks model, incorporating both observed and unobserved heterogeneity and allowing the heterogeneity terms for the various causes to be correlated. We employ a proportional hazard model with a piecewise constant baseline hazard.

Annex

Fig. 1.2. Map of Matlab showing villages of the HDSS area



Source: HDSS, Matlab, volume 35; Registration of health and demographic events 2002; Scientific report number 91.

Note: Government area is referred as comparison area in this thesis.

Table 1. *Timing of the Introduction of FPHSP Interventions in Matlab: 1977–89*

FPHSP interventions	Starting date	Treatment blocks A	B	C	D	Comparison areas
Family planning	October 1977	X	X	X	X	—
Basic MCH	January 1978	X	X	X	X	—
Tetanus toxoid to						
• Pregnant women	March 1978	X	X	X	X	—
• All women	December 1981	X	—	X	—	—
	December 1985	—	X	—	X	—
Oral rehydration therapy	January 1979	X	X	X	X	—
Measles vaccine (children aged 9–60 months)	March 1982	X	—	X	—	—
	December 1985	—	X	—	X	
Ante-natal care	September 1982	X	—	X	—	—
	January 1986	—	X	—	X	
Iron and folic acid to pregnant women	January 1985	X	—	X	—	—
	January 1986	—	X	—	X	
Oral cholera vaccine trial	May 1985	X	X	X	X	X
Vitamin A distribution (by government for children aged 6–60 months)	January 1986	X	X	X	X	X
EPI vaccinations: BCG, DPT and polio	March 1986	X	X	X	X	—
Maternity care	March 1987	—	—	X	X	—
Acute respiratory infections	April 1988	—	X	—	X	—
Nutritional rehabilitation	September 1988	X	X	X	X	—
Dysentery	April 1989	—	X	—	X	—

Source: Koenig and Strong, and Phillips *et al.* (*loc. cit.* in fn. 15). A non-FPHSP Extended Programme of Immunization was implemented in the comparison areas sometime between 1986 and 1989.

Note: The Table is copied from LeGrand and Phillips 1996.

Chapter 2

Infant death clustering in families: magnitude, causes, and the influence of better health services, Bangladesh 1982–2005[1]

2.1 Introduction

We report on an analysis of infant mortality in Bangladesh that focused on explaining the phenomenon of death clustering within families. The study used dynamic panel-data models that distinguished between two explanations of death clustering: (observed and unobserved) heterogeneity across families, and state dependence - a causal 'scarring' effect of the death of one infant on the survival chances on the next born child. Arulampalam and Bhalotra (2006, 2008) applied the logit version of this model to Indian data, while Omariba et al. (2008) used a similar probit model to analyse infant mortality in Kenya. Our study was the first to apply this type of model to Bangladesh.

Child mortality in Bangladesh remains an important issue. Under-five mortality declined sharply during the last decades of the previous century, but the reduction is levelling off and child mortality is not declining fast enough to meet Millennium Development Goal 4 of reducing under-five mortality by two-thirds between 1990 and 2015 (see United Nations 2001). Thus further reduction of child mortality remains a significant challenge. As in most developing countries, infant deaths (deaths before the age of twelve months) form the largest part of under-five mortality and therefore deserve special attention. Moreover, as for many other countries, data for Bangladesh reveal clear evidence of infant death clustering: the proportion of children who die in infancy is much larger among children whose previous sibling also died in infancy than among children whose sibling survived; see, for example, Swenson (1978), Koenig et al.

[1] This chapter is joint work with Arthur van Soest, Tilburg University and published as Saha and van Soest (2011). This paper has been presented in the Health & Labour Group seminar, Tilburg University, International Union for the Scientific Study of Population (IUSSP) 2009, Marrakesh, Morocco, and published in the Journal of Population Studies, November, 2011. We thank seminar participants, three anonymous referees and chief editor of Population Studies Journal for helpful suggestions and insightful comments.

(1990), Zenger (1993), Majumder et al. (1997), or Alam and David (1998). Understanding the causes of infant death clustering may give insight into how multiple deaths in a family could be prevented, and may thus contribute to the goal of reducing infant and child mortality.

Our study was based on prospective panel-data from 1982 to 2005 on mothers and children from the Matlab region, a rural area located 60 km southeast of Dhaka. Two sets of villages were covered: an intervention area with non-standard health services, and a (control) area with standard government-provided health care facilities. We expected the differences between the two areas to reveal the effects of additional health care services on infant mortality and death clustering.

2.2 Background

The large literature on the determinants of infant mortality in developing countries includes reports of studies from many countries showing that child deaths are clustered within families. See, for example, Das Gupta (1990) for India, Gubhaju (1985) for Nepal, Guo and Rodriguez (1992) and Guo (1993) for Guatemala, Curtis et al. (1993) and Sastry (1997) for Brazil, or Madise and Diamond (1995) for Malawi. Explanations of this phenomenon of death clustering are discussed extensively in, for example, Omariba et al. (2008, Section 2), and we summarize them only briefly here.

First, the clustering may be due to observed and unobserved characteristics of the mother, the family, or the local community; examples are adverse genetic traits, maternal health problems, inability to take care of the child, or environmental factors such as unsafe water supply or limited access to health care. All these factors may increase the risk for all children in a given family. It is also evident that death clustering is more pronounced among women of higher parity (Zaba and David 1996).

Second, death-clustering may be due to a causal effect of the death of one child on the survival chances of later siblings, an effect described as '(positive) scarring' (Arulampalam and Bhalotra 2006). One possible mechanism is that a child’s death leaves the mother depressed and that this affects the next child’s health in the womb or in infancy (on the 'depression hypothesis', see Steer et al. 1992 or Rahman et al. 2004). Another possible positive scarring mechanism is the 'replacement hypothesis': women whose child dies have their next birth sooner than they would have done otherwise, resulting in closely-spaced pregnancies that may lead to the health of the next born child being affected by nutritional depletion (see, e.g., Hobcraft et al. 1983). There might also be 'negative scarring' mechanisms': a reduced risk of infant death following the death of a sibling's death in infancy, owing to learning effects or to reduced competition for family resources (Alam 1995).

The older studies often attribute death clustering to socio-demographic covariates: either a causal scarring effect (the previous sibling’s survival status being included as a covariate), or unobserved family-level or community-level heterogeneity (with family-specific or community-

specific effects). A good example is Zenger (1993), who estimates models with either scarring or unobserved heterogeneity, but not both. Some studies include both unobserved heterogeneity and scarring (Guo 1993; Curtis et al. 1993; Sastry 1997; Bolstad and Manda 2001), but without accounting for the (bias induced by) potential correlation between the unobserved heterogeneity term and the previous child's survival-status dummy.

A major innovation was the study by Arulampalam and Bhalotra (2006), which explains infant mortality with an econometric dynamic panel-data model that at the same time also captures unobserved heterogeneity and the causal positive or negative scarring mechanisms (also referred to as 'state dependence' if panel data are used). Their model accounts for the endogeneity of previous sibling-survival status, thus avoiding the potential bias in previous studies. Arulampalam and Bhalotra (2006, 2008) applied this model to data for India and Omariba et al. (2008) used a similar model for Kenya. These studies all find positive scarring effects of varying sizes. For example, Arulampalam and Bhalotra (2008, Table 2) present separate estimates for 15 Indian states and find that, keeping other factors constant, infant death of the previous sibling increases the likelihood of infant death by between 2.2 percentage points (West Bengal and Punjab; two of the richest states) and 9.2 percentage points (Haryana). For Kenya, Omariba et al. (2008, p. 324) find a scarring effect of 4.8 percentage points. Since these estimates do not control for the length of the preceding birth interval, the estimated scarring effects include the effect through the birth interval (death of a child speeds up birth of the next child, and a shorter birth interval increases mortality risk). Arulampalam and Bhalotra (2008, Table 4) also present estimates with the preceding birth interval kept constant, which are estimates of the effects of scarring mechanisms that do not work through the birth interval. These estimates are still positive and in most cases significant, but 30 to 50 per cent smaller than the scarring effect, not controlling for preceding birth interval length. Bhalotra and van Soest (2008) extended this model by allowing birth intervals and fertility decisions to be endogenous to mortality. Keeping birth intervals constant, they find a scarring effect on neonatal mortality (death in the first month after birth) of 4.16 percentage points (p. 282).

The existing literature on child mortality includes a number of papers that focus on Bangladesh. A paper by Hale et al. (2006) used the same source of data that we used. They find that about 20 per cent of the differences in infant and child mortality between the intervention area and the comparison area can be explained by differences in reproductive behaviour (birth intervals, parity), and attribute the remaining part to differences in the quality of health services. In another paper, DaVanzo et al. (2007) analyse not only infant and child mortality but also stillbirths, miscarriages, and induced abortion. They conclude that mothers with a preceding non-live birth should receive counselling and monitoring. DaVanzo et al. (2008) find that shorter birth intervals are followed by higher mortality and conclude that some of their results are consistent with nutritional depletion of the mother and others with sibling competition. They also find support for mother-specific unobserved heterogeneity. They recommend that future research should use models that can disentangle these mechanisms. None of the preceding studies on Bangladesh use the dynamic panel-data models of Arulampalam and Bhalotra (2006, 2008) or

Omariba et al. (2008) to account properly for the various explanations of death clustering. Our study was directed at doing so.

Previous studies that used dynamic panel-data models are based on Demographic Health Surveys (DHS), for either India or Kenya. The samples were cross-sections of mothers who retrospectively reported their complete birth histories. Rich background information on, for example, the family's socio-economic status or the facilities in the area of residence was available, but only at the time of the survey. If these variables were used to explain infant mortality, the variables measured at the time of the survey were used as proxies for the same (unobserved) variables at the time of childbirth, which is why the existing studies used only background variables that were less likely to change over time. Our data were different: data were collected prospectively, avoiding possible recall error in birth and death histories and survival bias caused by mothers' mortality (Rosenblum 2009). Several variables were measured around the time of childbirth (like the father's occupational status and access to piped water; see also Section 3). Moreover, we knew whether women moved and could therefore select a sample of individuals who lived permanently in the same geographic area.

2.3 Data

2.3.1 Health and Demographic Surveillance System, Matlab

Since 1966, the International Centre for Diarrhoeal Disease Research, Bangladesh (ICDDR,B) has maintained a Health and Demographic Surveillance System (HDSS) in Matlab, a rural area located 60 km southeast of Dhaka. All the following data are recorded in this area for the complete population of about 220,000 people: births, deaths, causes of death, pregnancy histories, migration in and out of the area, marriages, divorces, and several indicators of socioeconomic status. The same data source has been used in many other studies of child mortality in Bangladesh, such as the studies of Bairagi et al. (1999), Majumder et al. (1997), Hale et al. (2006), Razzaque et al. (2007), and DaVanzo et al. (2007, 2008).

The ICDDR,B started the Maternal Child Health and Family Planning Programme (MCH-FP) project in October 1977 in half of the HDSS area, formerly known as the MCH-FP area and currently as the ICDDR,B area. In this half of the HDSS area additional health services were provided and additional data were collected on a range of health indicators. In the other half of the area, known as the comparison area, the usual programme of the Government of Bangladesh was maintained. Health and demographic data have been collected systematically in both halves of the HDSS area at regular household visits (every two weeks until January 1998 and once every month since then). In both halves, child mortality declined over time, though it was always smaller in the ICDDR,B area than in the comparison area. In 2005, the under-five mortality rates were 45.3 and 60.2 per thousand live births in the ICDDR,B area and the comparison area, respectively (ICDDR,B 2006). At each birth, the child is registered and the mother answers several questions about previous pregnancies. This yields the required information for all children whose births takes place in the HDSS area. If a woman migrates out

of the HDSS area, gives birth outside it, but migrates back within five years of the child being born, the child is still registered (birth date, survival status, etc.) as resident in the HDSS area. Otherwise, the child's records are not registered in HDSS and the information for the mother is incomplete. The child records make it possible to construct the sex (of each child born in the area), birth order, date of birth, and mother's age at birth.

In addition to monthly surveillance surveys, periodic surveys took place in 1982, 1996 and 2005 to collect socio-economic variables at the household and community level, such as source of drinking water, education and occupation of household members. Similar information was collected at marriage or migration into the HDSS area. We used the socio-economic information to construct several additional time-varying covariates at (approximately) the time of childbirth. The most important of these were source of drinking water (a dummy for access to piped water) and father's occupation. The change in access to piped water was especially substantial over time owing to the large-scale installation of piped water, initiated by the United Nations, after 1990.

2.3.2 Study sample

We combined the health and demographic surveillance system data from 70 villages in the ICDDR,B area and 79 villages in the comparison area, from 1 July 1982 until 31 December 2005 (the study period). Data from before 1 July 1982 are not (yet) available for research. The complete data set has records on about 63,000 mothers, with more than 165,000 child births – including live singleton births, multiple births, and stillbirths. For this study we eliminated mothers with multiple births as such children face much higher odds of dying, requiring a separate analysis, as documented in the demographic literature. We also deleted mothers with incomplete live birth information, usually due to migration out of Matlab during the period under study. Moreover, we discarded stillbirths. Finally, we excluded the children of three villages which shifted from the ICDDR,B area to the comparison area in 2000. This sample selection procedure leads to working samples of 31,968 children and 13,232 mothers in the ICDDR,B area and 32,366 children and 11,856 mothers in the comparison area.

2.3.3 Variables and descriptive statistics

The dependent variable infant death (y_{it}) was 1 if the child was observed to die before the age of 12 months and 0 otherwise. One of our main interests was in the effect of the lagged dependent variable y_{it-1}, the infant survival status of the preceding sibling. The other explanatory variables were included in x_{it}. They included birth order of the child (t), sex of the child, and age of mother at the time of birth of the child; education of the mother was captured by dummy variables for the level of education attained: no education (the omitted category), some primary education, or at least some secondary education. The mother's education level may have been a proxy for her ability to take good care of her children but may also have been a proxy for the family's socio-economic status. Education and occupation of the father also reflected the family's socio-economic status. The father's occupation was captured by a dummy for day labourers, a low socio-economic status occupation.

Following Arulampalam and Bhalotra (2006), birth intervals were not included in the main specification. Our estimates of the effect of scarring therefore included the potential effect through replacement—if infant death reduced the time until the next conception owing to a desire to replace the child that was lost, and a short birth interval increased the probability of infant death (see, e.g., Hobcraft et al. 1983), we could conclude that this was one mechanism that led to positive 'scarring'. In an alternative specification (Section 7), we added the preceding birth interval as a separate covariate. The mother's birth cohort also entered the model, giving insight into the trend of scarring over time. Another family-level covariate was religion: following Bhalotra et al. (2010a,b), who find that in Muslims in India have lower mortality probabilities than otherwise similar Hindus, we included a dummy for Muslims. More than 80 per cent of the mothers in our sample were Muslims, the others were mainly Hindus. To control for environmental factors, we included a dummy for access to running drinking-water (a dummy for piped drinking water/tubewell), and the distance to the nearest health facility (defined as a sub-centre or ICDDR,B hospital in the ICDDR,B area, or an Upazila Health Complex in the comparison area). (The health facilities offer emergency obstetric care, antenatal care, delivery, referral and contraceptive services, counselling on side effects of contraceptive use, and health education. In addition, children suffering from malnutrition and children with minor illnesses are treated, while children with severe illnesses are referred to a hospital.)

Table 1 presents sample means of the explanatory variables by area (percentages of outcome 1 for dummy variables). The average number of children born per mother is 2.42 in the ICDDR,B area and 2.73 in the comparison area; 19 per cent of families have more than three children in the ICDDR,B area, compared with 29 per cent in the comparison area (these figures are not reported in the Table). No differences between areas are observed in the mother's average age at birth. On average, mothers have lower education in the comparison area than in the ICDDR,B area. In the comparison area, mothers less often have access to the more hygienic sources of drinking water (tubewell/filter) and live much farther away from the nearest health facility (7.1 versus 1.9 kilometres).

In the ICDDR,B area, 5.09 per cent of all live births result in infant death; 10.66 per cent of all families experience at least one infant death and 0.79 per cent lose all their children in infancy. Infant mortality among first-borns (6.70 per cent) is substantially higher than among higher birth orders (3.95 per cent). In the comparison area, infant death is more common: 6.82 per cent of all children—8.90 per cent among first-borns; 5.62 per cent among higher birth orders. Of all families, 15.66 per cent experienced at least one infant death and 1.08 per cent lost all their children. Figure 1 presents the infant mortality rates by year of birth for both areas. It shows a decreasing trend in both areas until the late nineties. The infant mortality rate has always been higher in the comparison area than in the treatment area.

Table 2 shows the raw probabilities of infant death conditional on the survival status of the preceding sibling. Explaining this was one of the primary goals of this study. The probability of infant death is 4.34 percentage points (7.98 versus 3.64 per cent) if the preceding sibling dies as an infant in the ICDDR,B area, and 4.97 percentage points in the comparison area (10.14 versus 5.17 per cent). In other words, the likelihood of infant death is 2.2 (ICDDR,B area) or twice as high (comparison area) if the preceding sibling dies than if it survives.

2.4 Model specification

The econometric model we used, which is similar to the models used by Arulampalam and Bhalotra (2006, 2008) and Omariba et al. (2008), incorporates both scarring (state dependence) and the potentially confounding effects of unobserved inter-family heterogeneity. The model explains death during infancy of child *t* in family *i*. State dependence refers to whether the survival status of the previous child *(t-1)* in the same family (*i*) has an influence on the survival chances of the next child (*t*).

Let there be T_i children born alive in family *i* ($i=1, 2,...,N$ – the number of families or mothers in the sample); $t=1, 2,...,T_i$ denotes birth order. For $t>1$, the unobserved propensity to experience infant death y_{it}* is specified as

$$y_{it}^* = x'_{it}\beta + \gamma y_{it-1} + \alpha_i + u_{it}, \quad t=2,..., T_i \qquad (1)$$

The observed infant death outcome $y_{it} = 1$ if the child's propensity for death crosses a threshold normalized to zero, that is, if $y_{it}^* > 0$; otherwise, if $y_{it}^* \leq 0$, $y_{it} = 0$ and the child does not die in infancy. x_{it} is a vector of strictly exogenous observed explanatory variables and β a vector of coefficients; α_i captures unobserved heterogeneity at the mother level, which remains the same for all births of a given mother, accounting for all unobservable time-invariant characteristics influencing the child's propensity to die. The coefficient γ is associated with state dependence. (In principle child *t* could die in infancy before child *t-1* does, violating the sequence of events assumed in our model. This never happens in our data and is therefore ignored.) As in Omariba et al. (2008), the errors u_{it} are assumed to follow a standard normal distribution, independent of each other and of x_{is}, $s=1,...,T_i$. In robustness checks in Section 7, we also report results with logistic errors (following Arulampalam and Bhalotra 2006, 2008).

The model assumes that the history of infant deaths among older children other than the immediately preceding child has no direct effect on y_{it}*. For example, if child *t-2* died in infancy, in our model this will affect the risk of death of child *t-1* and, thereby, also the risk of death of child *t*, but there is no *direct* effect on death of child *t*. This is the first-order Markov assumption (Zenger 1993; Arulampalam and Bhalotra 2006).

With the above specification, the conditional probability of death for infant *t* of mother *i*, given y_{it-1}, x_{it}, and α_i, is given by:

$$P[y_{it}=1 | y_{it-1}, x_{it}, \alpha_i] = \Phi[(x'_{it}\beta + \gamma y_{it-1} + \alpha_i)], \qquad (2)$$

where Φ denotes the standard normal cumulative density. The joint conditional probability of the observed sequence of binary outcomes is given by:

$$P(y_{i1,\ldots\ldots\ldots,} y_{i,T(i)} \mid \alpha_i, x_{i1}, \ldots, x_{iT(i)})$$

$$= P(y_{iT(i)} | y_{iT(i)-1,} \alpha_i, x_{iT(i)})$$

$$\times P(y_{i,T(i)-1} | y_{i,T(i)-2,} \alpha_i, x_{i,T(i)-1})$$

$$\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots$$

$$\times P(y_{i2} | y_{i1,} \alpha_i, x_{i2}) \, P(y_{i1} | \alpha_i, x_{i1}) \qquad (3)$$

Using the sequence above requires specifying $P(y_{i1}|\alpha_i,x_{i1})$ (the 'initial condition problem' in dynamic models with unobserved heterogeneity; see Heckman, 1981; alternative approaches are compared in a simulation study by Arulampalam and Stewart 2009 who conclude that the various estimators perform similarly well).

Modelling the outcome of the first child is especially relevant because the first child shares unobservable traits α_i with its younger siblings. Without unobserved heterogeneity ($\alpha_i=0$ for all i), the initial observation could be treated as exogenous, and equation (1) could be estimated using the sample of second and further children only. Since the correlation between α_i and y_{it-1} that makes y_{it-1} endogenous in equation (1) is probably positive, ignoring it would probably lead to overestimation of γ (Fotouhi, 2005). This is why we specify a separate equation for mortality of the first-born child. This equation has the same form as for equation (1) and is given by

$$y_{i1}^* = x'_{i1}\pi + \theta\alpha_i + u_{i1} \qquad (4)$$

with the same assumptions for the error term u_{i1} as for the other u_{it}. The auxiliary parameters π and θ are estimated jointly with the parameters of interest. Exogeneity of first-child survival corresponds to $\theta=0$, which can be tested in a standard way. Equation (4) implies that the conditional probability of infant death for the first-born child is given by:

$$P(y_{i1}=1|\alpha_i, x_{i1}) = \Phi\,[x'_{i1}\pi + \theta\alpha_i] \qquad (5)$$

Combining equations (1) and (4) gives the following conditional probability of an observed sequence of binary outcomes $y_{i1,\ldots,}\, y_{iT(i)}$ for all children of family i:

$$P(y_{i1}y_{i2,\ldots}y_{iT(i)} \,|x_{iT(i)}, \ldots, x_{i1,}\, \alpha_i) =$$

$$\Phi\{(x'_{i1}\pi + \theta\alpha_i)(2y_{i1}-1)\}\Pi_{t=2,\ldots,T(i)}\{\Phi(x'_{it}\beta+\gamma y_{it-1}+\alpha_i)(2y_{it}-1)\} \qquad (6)$$

We assume that α_i is normally distributed with mean 0 and variance σ_α^2, independent of all x_{it} and u_{it}, $t=1,\ldots,T_i$. The likelihood contribution for family *i* is then given by

$$L_i = \int P\,(y_{i1}, y_{i2,..}, y_{iT(i)} | x_{iT(i)}, \ldots, x_{i1,} \alpha) f(\alpha)\, d\alpha \qquad (7)$$

Where $f(\alpha)$ is the density of $N(0,\sigma_\alpha^2)$. The integral in (7) can be computed numerically using Gauss-Hermite quadrature (Butler and Moffitt 1982). We used the Stata code of Stewart (2007) to obtain the maximum likelihood estimates, based on 32 quadrature points.

2.5 Estimation results

The estimates of several versions of the model are presented in Table 3a (equation (1), for birth orders larger than 1) and Table 3b (equation (4), for first-borns). In Model 1, the only explanatory variable is infant survival status of the previous sibling (y_{it-1}); Model 2 adds child and mother-level factors, and Model 3 also adds environmental and father-specific factors. We estimated several model specifications, including models with dummies for birth order and mother's age at birth, and present the models that gave the best fit to the data, which is why some background variables enter as dummies and others as continuous variables.

The estimates of γ for Model 1 imply that the death of the preceding sibling has a positive significant effect on the probability of infant death in the comparison area, whereas a positive but insignificant effect is found in the ICDDR,B area.

The partial effect of y_{it-1} on $P[y_{it}=1 | y_{it-1,} x_{it,} \alpha_i]$ can be derived from the estimates by constructing counterfactual outcome probabilities $p_{0,} p_{1,}$ fixing y_{it-1} at 0 and 1, evaluated at the overall means of the exogenous variables and for $\alpha_i = 0$; the difference between p_1 and p_0 can be interpreted as the average partial effect (APE); the ratio p_1/p_0 is the predicted probability ratio (PPR) (Stewart 2007, p.522). Both are indicators of state dependence. In Model 1, the APE is 2.12 percentage points in the comparison area and 0.42 percentage points in the ICDDR,B area (see Table 4). In terms of PPR, the likelihood of infant death is about 41 per cent greater if the older sibling dies at infancy in the comparison area and about 12 per cent in the ICDDR,B area.

In the comparison area, including child and mother-level variables reduces the estimate of γ and its significance level (Model 2); adding the father's characteristics (Model 3) leads to a small increase of γ and its significance level. In the ICDDR,B area, adding the regressors in Models 2 and 3 leads to small negative and insignificant estimates of the scarring effect. In fact, in the ICDDR,B area all three models find an insignificant scarring effect. Apparently, the positive correlation between survival of consecutive children in the raw data (Table 2, lines 5 and 6) can be explained by observed and unobserved heterogeneity, leaving no significant role for scarring. This may also mean that positive and negative scarring effects eliminate each other.

The predicted probability ratios (PPR) in Table 4 show that according to Model 3, the likelihood of infant death in the comparison area is 29 per cent higher if the previous child dies at infancy than if it survives. This effect is smaller than the estimate of Model 1, owing to the

inclusion of the covariates. Comparing the estimated scarring effect of 1.52 percentage points with the differential of 4.97 percentage points in the raw data shows that in the comparison area, scarring explains about one third of within-family clustering of infant deaths. The remaining part is explained by observed and unobserved heterogeneity.

The estimates of θ in Table 3b can be used to test whether the initial outcome (infant survival of the first child) can be treated as exogenous. If θ=0, the unobservables in equation (4) would be uncorrelated with the unobservables in the main equation (1) and there would be no need to estimate jointly the main equation (1) and the equation for the initial outcome (equation (4)) (see Stewart, 2007, or Arulampalam and Bhalotra, 2006). The null hypothesis θ=0 is firmly rejected for all our models in both areas. This confirms the importance of accounting for the initial condition.

In Model 3, the proportion of the total unsystematic variance that is attributable to family-level unobservables α_i is estimated to be 8.1 per cent in the comparison area and 22.2 per cent in the ICDDR,B area (Table 3b). The null hypothesis of no family-level unobservables is decisively rejected in both areas for all models, but unobserved heterogeneity plays a much larger role in the ICDDR,B area than in the comparison area.

The other covariates often play different roles in the two areas and for first-born and non-first-born children. Among first-born children, sons are more likely to die than daughters in both areas, but the difference in the comparison area is smaller than in the ICDDR,B area. No significant sex differences are observed for higher-birth orders. This is consistent with the results of a study by Waldron (1983), who finds that infant mortality is inherently larger for boys than for girls, but that this outcome can be reversed by environmental disadvantages for female children. These environmental factors may be reduced by the extensive health services in the ICDDR,B area.

In the ICDDR,B area, the probability of infant death has a U-shaped relationship with the mother's age at the time of childbirth, with a minimum at about age 30. In the comparison area, the pattern is similar for first-born children but there is no evidence of increasing death probabilities at older ages for higher-birth orders. The mother's birth-cohort dummies indicate significantly lower infant mortality probabilities for younger cohorts in both areas for first-born and higher birth orders. This is probably because of a time trend in hygienic circumstances and health technology, in line with the declining trends in Figure 1 and the findings of Arulampalam and Bhalotra (2008). On the other hand, Omariba et al. (2008) found no significant effect of the mother's birth cohort.

In both areas, education of the mother significantly reduces the risk of infant mortality for the first child, but not for higher birth orders once the father's education is also controlled for (Model 3). On the other hand, education of the father significantly reduces infant mortality of higher birth orders but not of first-born children. Both education variables are measures of the family's socio-economic status, and the general conclusion is that higher socio-economic status

implies lower mortality. The third indicator of (low) socio-economic status is a dummy indicating whether the father is a day labourer. As expected, it has a significantly positive effect on mortality for higher birth orders, a finding similar to that of D'Souza and Bhuiya (1982); this may reflect the association between high mortality and poor socio-economic conditions with insecure household income. Mosley and Chen (1984) also relate this to the stable availability of a basic minimum food supply of a variety sufficient to ensure adequate amounts of nutrients.

Those who used tubewell or pipe water as a source of drinking water are less likely to see their children die in infancy, but this finding is significant for higher birth orders in the ICDDR,B area only. The distance to the nearest health facility has a significantly positive effect on infant mortality in the comparison area, and the effect is particularly pronounced for first-born children. That no significant effect is found in the ICDDR,B area may be due to the fact that almost all families live rather close to a health facility in that area.

A formal way to compare the results in the ICDDR,B area and the comparison area is presented in the Annex to this chapter. Here the differential in mortality between the two areas is decomposed into a part that can be ascribed to differences in the distribution of the covariates and a residual part (ascribed to differences in parameters).

2.6 Alternative specifications and robustness checks

To analyse the sensitivity of our results to several specification choices, we estimated several alternative models. Table 5 summarizes the main results, focusing on the estimates of the scarring effect (γ). The first row reproduces the results from our benchmark model, Model 3 in the previous section. To show the (upward) bias on the scarring effect when unobserved heterogeneity is discarded, Row 2 presents the results of a simplified model without unobserved heterogeneity. As expected, this leads to much higher estimates of the scarring effect: ignoring unobserved heterogeneity implies that infant death clustering owing to unobserved heterogeneity will incorrectly be attributed to scarring. This is exactly consistent with the explanation of Arulampalam and Bhalotra (2006) for the necessity of incorporating both unobserved heterogeneity and scarring in the same model (see also Section 2).

Row 3 adds the log birth interval as an additional regressor to the benchmark model. It has a strong and significant negative effect on mortality, consistent with the existing literature. Adding it leads to a smaller estimate of the scarring effect, which now has a different interpretation: since birth intervals are kept constant, the scarring effect no longer includes the (positive) effect through replacement and depletion (see Section 2). In other words, the difference between the scarring effects in the benchmark model and the effects of the model with the birth interval can be interpreted as an estimate of the effect through the mechanism that infant mortality speeds up the next birth, and a faster next birth leads to a higher mortality risk. For the comparison area, the estimated scarring effect in row 3 is virtually zero, so that the complete scarring effect in the benchmark model works through the birth interval. For the ICDDR,B area,

the effect in row 3 is significantly negative, suggesting an effect of sibling competition or learning (see Section 2).

Since Arulampalam and Bhalotra (2006, 2008) used logistic errors while Omariba et al. (2008) used standard normal errors, we wanted to investigate the sensitivity of the results to this choice. Row 4 replaces the normally distributed errors in the benchmark model by logistic errors. The parameter estimates change owing to a different normalization (the variance of the error terms is now $\pi^2/3$ instead of 1), but significance levels and marginal effects are similar to those in the benchmark model. This also applies to the other parameters of the model (details available on request). Still, the estimated scarring parameter in the comparison area changes from marginally significant (at the 5 per cent level) to marginally insignificant (*p*-value 0.062). In terms of log likelihood, the probit model fits slightly better than the logit model, which is why we used probit as the benchmark.

Finally, to obtain more efficient estimates, we combined the samples for the two areas, tested whether the coefficients in the two areas were significantly different, and imposed equality for sets of coefficients where supported by the test. This gives a more parsimonious version of the model (which is not rejected against the benchmark model by a likelihood ratio test). The final row of the table shows that the efficiency gain in the estimates of the scarring parameter (which is significantly different in the two areas) is quite small. The standard errors and point estimates hardly change compared to the benchmark.

In additional estimations, we also investigated interactions of the scarring effect with other covariates (results not reported in the Table), but this did not lead to additional insights. For example, interactions of the dummy for infant mortality of the previous child with educational dummies were all insignificant, thus offering no evidence that scarring effects differ by education level.

2.7 Discussion and conclusion

We analysed the determinants of infant mortality in Bangladesh in areas with and without health services beyond the standard services provided by the government. We used recently developed methods to account for heterogeneity across families as well as state dependence in infant mortality. Our methods thus accounted for competing explanations for the stylized fact that a child has a higher probability of death if the previous child of the same mother had died. Separating the causal effect from unobserved heterogeneity has important implications for policy in this area and for research on the inter-relationships of family behaviour and mortality. Indeed, we find that controlling for unobserved heterogeneity is necessary to prevent substantial overestimation of the causal 'scarring' effect of the death in infancy of the previous child.

We find that in the comparison area, the likelihood of infant death is almost 29 per cent greater if the older sibling dies in infancy than if it survives. Adding the birth interval to the model suggests that this scarring effect can be fully attributed to a mechanism that works through

birth intervals: infant death leads to a shorter next birth interval (*replacement*) and a shorter birth interval increases mortality risk (*nutritional depletion).* In the ICDDR,B area, the same mechanism plays a similar but smaller role, perhaps because of better health services and more information on contraceptives and health risks. Moreover, it is offset by an effect of *learning* or *sibling competition* in the opposite direction. The difference between scarring effects in the two areas is consistent with the finding of Arulampalam and Bhalotra (2008) that (positive) scarring is weaker in more developed regions.

Unobserved time-persistent heterogeneity among mothers captures 22 per cent of the total unsystematic variation in infant deaths in the ICDDR,B area, compared to only 8 per cent in the comparison area. An explanation may be that some mothers who receive health information are better at exploiting this than others, so that additional health information increases heterogeneity. Another explanation might be that the ICDDR,B area is divided into four sub-regions ('blocks'), with interventions such as vaccinations phased out at different times in different blocks, so that different children benefit differently from these interventions; dummies for whether specific interventions were introduced at the time of birth were not significant, however, so that this explanation could not be confirmed. Echoing the results of Hale et al. (2006), we do find that the effect of the mother's birth cohort is stronger in the ICDDR,B area than in the comparison area, possibly reflecting the advantages of introducing extensive health services in the ICDDR,B area.

Estimating the model for the higher educated mothers only (results not reported) suggests that the mother-specific variation in infant deaths is 16 per cent greater among mothers with secondary or higher education than for the complete ICDDR,B sample. This finding confirms the prediction that "the new interventions will tend to increase the inequality since they will initially reach those who are already better off" (Victora et al. 2001; Razzaque et al. 2007). On the other hand, in the comparison area, greater unobserved heterogeneity is observed among mothers with no education level, possibly owing to variation in innate ability (Das Gupta 1990).

Our findings confirm the general result that low socio-economic status increases the risk of infant death, but we find some remarkable and policy-relevant differences between first-born and later-born children. For the first-born, the mother's education seems particularly important, suggesting that it is particularly difficult for low educated first-time mothers to create a healthy environment for a newborn child. At the time of first birth, education may improve the women's autonomy and decision making; a woman's autonomy is lowest when she is a young mother, and education helps women to overcome the barriers imposed by low autonomy in a traditional society (Das Gupta 1990). For higher birth orders where competition among siblings for scarce resources matters, the father's education seems more important, possibly as an indicator of the family's general socio-economic status. A longer distance to the nearest health facility leads to higher mortality in the comparison area and this effect is more pronounced for the first-born child than for children of higher birth order, possibly reflecting the social taboos restricting the mobility of younger mothers. The effect of safe drinking water in the ICDDR,B area—a finding

unique among studies of death clustering—is consistent with the discussion in the background section.

We believe our findings can contribute to the formulation of effective policies targeted at achieving the fourth millennium development goal of reducing under-five mortality. The differences between the two areas highlight the important role of extensive maternal and child health interventions: the kind of extensive health services and health information available in the ICDDR,B area. Of particular note is the fact that in the ICDDR,B area we find some families that appear to have learnt from the experience of infant death in the past how to reduce the probability of the death of the next child. On the other hand, the finding that unobserved heterogeneity in the ICDDR,B area is much greater than in the comparison area implies that not everyone benefits equally from the health interventions, which suggests that policies that increase equity in the use of interventions may help to reduce infant mortality further.

Tables

Table 1. Descriptive statistics used in a study of infant mortality, Matlab, Bangladesh 1982–2005.

Variables	ICDDR,B area	Comparison area
Infant deaths (all live-births) (%)	5.09	6.82
Infant deaths excluding first-borns (%)	3.95	5.62
Infant deaths among first borns (%)	6.70	8.90
Families with no infant deaths (%)	89.34	84.34
Families in which all births die in infancy (%)	0.79	1.08
Age of mother at first birth[1]	21.16 (3.23)	21.08 (3.21)
Age of mother at birth[1]	24.70 (5.03)	24.58 (4.85)
Mother's education level (%):		
No education	48.48	50.50
Some primary education	24.86	25.51
At least some secondary education	26.66	23.99
Mother Muslim (%)	82.71	89.85
Child male (%)	50.97	51.12
Birth order (%)		
1	41.39	36.63
2	28.93	26.74
3	17.62	18.26
4+	12.06	18.36
Father's education level (%):		
No education	55.67	56.28
Some primary education	22.65	24.15
At least some secondary education	21.68	19.57
Father day labourer (%)	19.61	20.96
Drinking water tubewell/piped water (%)	87.76	76.91
Distance to nearest health centre (km)[1]	1.87 (0.98)	7.07 (4.04)
Number of mothers in sample	13,232	11,856
Number of children in sample	31,968	32,366

[1]Means and (in parentheses) standard deviations.

Source: Matlab DSS data

Table 2. Clustering and scarring in sibling infants deaths: Raw probabilities of infant deaths conditional on the survival status of previous sibling, Bangladesh 1982–2005.

	ICDDR,B area	Comparison area
1 Infant death/1000 live births	50.1	68.2
2 Infant death/1000 live births, no first borns	39.5	56.2
3 Probability ($y_{it} = 1 \mid y_{it-1}=1$), p_1	0.0798	0.1014
4 Probability ($y_{it} = 1 \mid y_{it-1}=0$), p_0	0.0364	0.0517
5 Persistence due to y_{it-1} (difference measure)	0.0434	0.0497
6 Persistence due to y_{it-1} (ratio measure)	2.192	1.961

Notes:

This table is built up in a way similar to that of panel A of Table 2 in Arulampalam and Bhalotra (2006).

In rows 3 and 4, p_1 is the observed fraction of infant deaths among those whose previous sibling died at infancy; p_0 is the fraction of infant deaths among those whose previous sibling survived at infancy.

APE: average partial effect; PPR: predicted probability ratio.

Table 3a. Estimation results of dynamic random–effects probit models for death in infancy, birth order > 1, Bangladesh 1982-2005.

Covariates	ICDDR,B area			Comparison area		
	Model 1	**Model 2**	**Model 3**	**Model 1**	**Model 2**	**Model 3**
Previous sibling died (γ)	0.0553	-0.0840	-0.0920	0.1830	0.1236	0.1323
	(0.0761)	(0.0843)	(0.0845)	(0.0572)	(0.0615)	(0.0615)
Male		0.0385	0.0354		0.0140	0.0135
		(0.0380)	(0.0381)		(0.0302)	(0.0302)
Birth order		0.0864	0.0956		-0.1183	-0.1119
		(0.0988)	(0.0988)		(0.0540)	(0.0539)
Birth order squared		-0.0149	-0.0152		0.0156	0.0153
		(0.0133)	(0.0133)		(0.0064)	(0.0064)
Mother's age at birth		-0.1768	-0.1690		-0.0671	-0.0615
		(0.0350)	(0.0028)		(0.0334)	(0.0336)
Mother's age at birth squared		0.0028	0.0028		0.0008	0.0007
		(0.0006)	(0.0006)		(0.0006)	(0.0006)
Muslim		-0.0621	-0.0233		-0.0866	-0.0647
		(0.0525)	(0.0549)		(0.0490)	(0.0507)
Mother's education: some primary		-0.0973	-0.0559		-0.0289	0.0104
		(0.0499)	(0.0515)		(0.0387)	(0.0399)
Mother's education: at least some secondary		-0.1752	-0.0489		-0.1629	-0.0810
		(0.0611)	(0.0668)		(0.0513)	(0.0550)
Mother's birth cohort:						
1966-70		-0.0318	-0.0114		-0.1638	-0.1531
		(0.0522)	(0.0528)		(0.0398)	(0.0405)
1971-75		-0.1648	-0.1398		-0.3027	-0.3010
		(0.0613)	(0.0625)		(0.0470)	(0.0487)
1976+		-0.2059	-0.1725		-0.5552	-0.5468
		(0.0714)	(0.0731)		(0.0596)	(0.0621)
Father's education: some primary			0.0428			-0.0473
			(0.0491)			(0.0387)
Father's education: at least some secondary			-0.2058			-0.1473
			(0.0649)			(0.0491)
Father's occupation is			0.1275			0.0876
			(0.0516)			(0.0392)
Source of drinking water: tubewell / piped water			-0.1858			-0.0211
			(0.0597)			(0.0396)
Distance to health facility (km)			-0.0021			0.0079
			(0.0210)			(0.0039)
Constant	-1.9674	0.6794	0.5714	-1.7106	0.0224	-0.1564
	(0.0461)	(0.4721)	(0.4798)	(0.0272)	(0.4472)	(0.4540)

Notes: Standard errors are in parentheses.

Reference categories of categorical variables used in the model: female, non-Muslim, no educational degree, no access to piped water, not day labourer, mother born before 1966.

Table 3b. Estimation results of dynamic random-effects probit models for death in infancy, first-born children, Bangladesh 1982–2005.

Covariates	ICDDR,B area			Comparison area		
	Model 1	**Model 2**	**Model 3**	**Model 1**	**Model 2**	**Model 3**
Male		0.1277	0.1277		0.0666	0.0636
		(0.0372)	(0.0371)		(0.0341)	(0.0341)
Mother's age at birth		-0.0828	-0.0812		-0.1544	-0.1524
		(0.0335)	(0.0334)		(0.0356)	(0.0355)
Mother's age at birth squared		0.0013	0.0013		0.0028	0.0028
		(0.0007)	(0.0007)		(0.0008)	(0.0007)
Muslim		-0.0119	-0.0009		-0.0387	-0.0078
		(0.0478)	(0.0496)		(0.0556)	(0.0571)
Mother's education: some primary		-0.2055	-0.1953		-0.1593	-0.1370
		(0.0479)	(0.0452)		(0.0441)	(0.0445)
Mother's education: at least some secondary		-0.3527	-0.3045		-0.3355	-0.3048
		(0.0510)	(0.0563)		(0.0482)	(0.0526)
Mother's birth cohort:						
1966-70		-0.1330	-0.1290		0.0009	0.0158
		(0.0571)	(0.0573)		(0.0584)	(0.0567)
1971-75		-0.1847	-0.1771		0.0087	0.0298
		(0.0609)	(0.0610)		(0.0585)	(0.0612)
1976+		-0.4174	-0.4087		-0.1944	-0.1352
		(0.0609)	(0.0631)		(0.0588)	(0.0637)
Father's education: some primary			0.0606			-0.0286
			(0.0461)			(0.0426)
Father's education: at least some secondary			-0.0798			-0.0002
			(0.0535)			(0.0485)
Father's occupation is day labourer			0.0302			0.0893
			(0.0449)			(0.0418)
Source of drinking water: tubewell / piped water			-0.0379			-0.0650
			(0.0518)			(0.0423)
Distance to health facility (km)			0.0147			0.0154
			(0.0182)			(0.0042)
Constant	-1.6381	0.1205	-0.1836	-1.3946	0.7929	0.5771
	(0.0545)	(0.3994)	(0.4054)	(0.0325)	(0.4236)	(0.4270)
ρ	0.1682	0.2245	0.2221	0.0941	0.0897	0.0812
	(0.0365)	(0.0411)	(0.0417)	(0.0238)	(0.0276)	(0.0276)
θ **(see eq. 4)**	0.9808	0.7562	0.7476	0.8317	0.8103	0.8045
	(0.2558)	(0.1717)	(0.1747)	(0.2866)	(0.3177)	(0.3483)
Log-likelihood	-6320	-6172	-6076	-7953	-7775	-7755

Note:

$\rho = \frac{\sigma_\alpha^2}{\sigma_\alpha^2 + \sigma_u^2}$ is the proportion of the total unsystematic variance attributed to the family-level unobservables α_i

Table 4. Average predicted probabilities of infant death given previous sibling's survival status, Bangladesh 1982–2005.

	ICDDR,B area			Comparison area		
	Model 1	**Model 2**	**Model3**	**Model 1**	**Model 2**	**Model 3**
P_1	0.0406	0.0335	0.0321	0.0730	0.0678	0.0681
P_0	0.0364	0.0394	0.0384	0.0518	0.0536	0.0530
APE: $p_1 - p_0$	0.0042	-0.0059	-0.0063	0.0212	0.0141	0.0152
PPR: p_1/p_0	1.116	0.850	0.836	1.410	1.263	1.286

Notes: p_1 is computed using the estimated marginal predicted probability of y_{it} for each observation under the condition that the previous sibling died ($y_{it-1} = 1$), averaged over all observations (with $t > 1$). Similarly, p_0 is obtained setting $y_{it-1} = 0$.

APE: average partial effect; PPR, predicted probability ratio.

Table 5. Scarring parameter (γ) and log likelihoods of alternative specifications of models estimating infant mortality, Bangladesh 1982–2005.

	ICDDR,B area		Comparison area	
	Estimate of γ (standard error)	**Log likelihood**	**Estimate of γ (standard error)**	**Log likelihood**
Benchmark model (Table 3, Model 3)	-0.092 (0.084)	-6153.10	0.132* (0.061)	-7755.52
No unobserved heterogeneity	0.296** (0.051)	-6173.83	0.276** (0.044)	-7757.00
With log birth interval	-0.181* (0.082)	-6145.42	-0.009 (0.069)	-7742.60
Logit model	-0.140 (0.151)	-6153.11	0.230 (0.123)	-7757.88
Joint estimation both areas	-0.037 (0.074)	-13,922.75+	0.121 (0.062)	

Notes:

*p–value < 0.05; ** p–value < 0.01

+: log likelihood of joint model, imposing equality of 22 slope coefficients in the two areas.

Figure

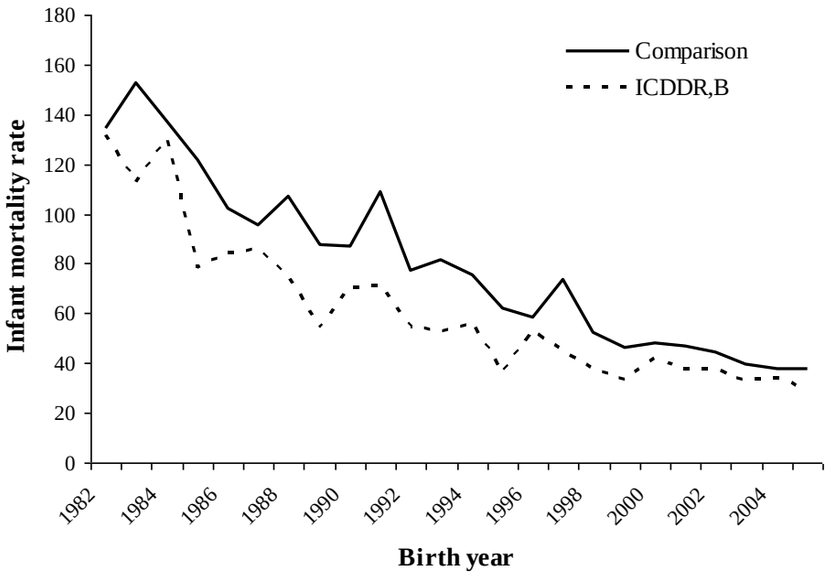


Figure 1. Infant mortality rates (per 1,000 live births) by birth year, ICDDR,B area and comparison area, Bangladesh 1982–2005

Source: Matlab DSS data.

Annex

Decomposing the Difference between Areas

The aggregate prediction of the infant mortality rate according to model 3 for all children (first born as well as others) is about 49 per thousand live births in the ICDDR,B area and 67 per thousand live births in the comparison area, a difference of 18 per thousand live births. We analyze the gap between the two areas using the common technique of decomposing differences in mean levels into those due to different observable characteristics or "endowments" and those due to different effects of characteristics or "coefficients" (Blinder 1973; Oaxaca 1973). In the standard case of a linear model the technique requires coefficient estimates from linear regressions for the outcome of interest and sample means of the independent variables used in the regressions. Adjustments for the case of a nonlinear model such as our binary choice model were introduced by Fairlie (2005) and Yun (2004).

Here we follow the decomposition methodology proposed by Yun (2004) for the probit model (binary dependent variable), which can straightforwardly be extended to the dynamics random effects probit model taking into account of the initials conditions and the unobserved heterogeneity. The 'aggregate' or 'overall' mean difference in infant mortality between the two areas ICDDR,B (group A) and comparison (group B) can be decomposed as follows:

$$\begin{aligned}\bar{Y}_A - \bar{Y}_B = &\left[\overline{\Phi(X_A\beta_A + \lambda_A\alpha_A)} - \overline{\Phi(X_B\beta_A + \lambda_A\alpha_B)}\right] + \\ &+\left[\overline{\Phi(X_B\beta_A + \lambda_A\alpha_B)} - \overline{\Phi(X_B\beta_B + \lambda_B\alpha_B)}\right]\end{aligned} \qquad (1)$$

The means are taken over either all first born children or all higher birth orders of all mothers in each area and over the random effects. We have used shorthand notation, dropping indexes i and t and combining expressions for the first born child and the second and higher birth orders. For example, X_A includes x_{it} as well as y_{it-1} for *t>1*, β_A denotes either π (for birth order 1) or (β,γ) (for higher birth orders), and $\lambda = \theta$ or $\lambda = 1$ for birth order equal to 1 or larger than 1, respectively.

The first component in the decomposition in equation (1) is the "endowment" or "composition effect", the part of the difference explained by differences in (observed and unobserved) characteristics of in the two samples. The second is the residual difference keeping characteristics constant. To estimate the two components, we replace the parameters by the estimates for Model 3 in Table 3 for the treatment area (*A*) or the comparison area (*B*). This is referred to as decomposition 1. We also present the results in the reverse order, i.e., taking the comparison area as group A and the treatment area as group B (and adding minus signs for

comparability), which we refer to as decomposition 2. The unobserved heterogeneity terms are replaced by random draws from their estimated normal distributions.

To understand which characteristics contribute to explaining the mortality difference between the two regions, we also performed the so-called detailed decomposition, again following Yun (2004). For this purpose, equation (1) is rewritten as follows:

$$\begin{aligned}\bar{Y}_A - \bar{Y}_B = &\sum_{i=1}^{i=k} W_{\Delta x}^{i}\left[\overline{\Phi(X_A\beta_A + \lambda_A\alpha_A)} - \overline{\Phi(X_B\beta_A + \lambda_A\alpha_B)}\right] \\ &+\sum_{i=1}^{i=k} W_{\Delta \beta}^{i}\left[\overline{\Phi(X_B\beta_A + \lambda_A\alpha_B)} - \overline{\Phi(X_B\beta_B + \lambda_B\alpha_B)}\right]\end{aligned} \qquad (2)$$

Where the "weights" are given by

$$W_{\Delta X}^{i} = \frac{\left(\overline{X}_A^{i} - \overline{X}_B^{i}\right)\beta_A^{i}}{\left(\overline{X_A} - \overline{X_B}\right)\beta_A} \text{ and } W_{\Delta \beta}^{i} = \frac{\left(\beta_A^{i} - \beta_B^{i}\right)\overline{X_B^{i}}}{\left(\beta_A - \beta_B\right)\overline{X_B}}, \text{ so that } \sum_{i=1}^{i=k} W_{\Delta x}^{i} = \sum_{i=1}^{i=k} W_{\Delta \beta}^{i} = 1.$$

We focus on the contribution of each variable to the endowment effect, the first part in (2). We present the results for the second part for completeness, but we do not have a good interpretation for these in our context.

The bottom rows of the two panels in Table A give the results of the overall decomposition (for Model 3) ("Total"). For the first born child, almost two thirds of the mortality gap is explained by characteristics according to both decomposition 1 and decomposition 2 (16.3 or 17.0 per one thousand live births, of a total gap of 24.9 per thousand live births). The detailed composition shows that this is almost completely due to the variable *distance to the nearest health facility.* This variable has a strong (negative) effect on survival chances and the distances are much larger in the comparison area than in the treatment area.

For higher birth orders, differences in characteristics explain a smaller part of the total gap and the results are sensitive to which of the two decompositions is used. According to decomposition 2, the endowment effect is about one third of the total effect (5 out of the total gap of 15 per thousand live births) and again this is mainly driven by the distance to the nearest health facility, though mother's age at birth also plays a role: mortality falls with age of the mother at birth, and mothers in the treatment area are somewhat older, on average. According to decomposition 1, however, the difference in distance to a health facility hardly plays a role. The reason is that this is now weighted by the coefficient estimate for the distance variable in the treatment area, which is small and insignificant. Accordingly, decomposition 1 also attributes a much smaller contribution to all observed differences in characteristics (1.5 out of 15).

Chapter 3

Reproductive Behaviour and Infant Deaths: Causal Analysis[2]

3.1 Introduction

According to the demographic transition theory, there is a strong correlation between childhood mortality and fertility. Empirical evidence has shown that a decline in childhood mortality is often a prerequisite for fertility decline (Chowdhury et al. 1976; Matthiesson and McCann 1978; Pritchett 1994; Wolpin 1997). Other studies have emphasized the reverse direction of this causation, e.g., high fertility and the close birth-spacing associated with it cause an increase in child mortality (Cleland and Sathar 1984; Curtis et al. 1993). Yet another set of studies emphasized that the analysis of the direction of causality with birth interval data is hampered by the close interrelations between child mortality and fertility (Zimmer 1979; Santow and Bracher 1984).

Studies have emphasized that the observed associations between child mortality, birth spacing, and fertility can be due to various causal mechanisms, but may also be explained by common unobserved factors that drive the various processes. From the point of view of policies aimed at optimal birth spacing, reducing mortality, and reducing fertility, it is crucial to identify the importance of the various causal mechanisms and alternative explanations. If associations reflect spurious correlation or reverse causation, then the implications for policy design can be dramatically altered (Moffitt 2005). Ben-Porath (1976, p. S168) already argued that in micro data, associations may not reflect causal effects but may be spurious and reflect left-out variables operating simultaneously on fertility and mortality. In addition, a recent study conducted by DaVanzo and her colleagues (DaVanzo et al. 2008) in Matlab emphasized the importance for joint analysis including interval lengths and mortality, allowing for correlated risks among different births to the same mother.

To achieve this latter goal, we use a panel data model similar to the one introduced by Bhalotra and van Soest (2008). This model incorporates various causal mechanisms as well as

[2] This chapter is joint work with Arthur van Soest, Tilburg University. This paper has been presented in the Statistics and Econometrics seminar, Tilburg University. We thank seminar participants and members of the committee for helpful comments and suggestions.

unobserved heterogeneity, exploiting the sequence of all births and deaths to a mother to accommodate the correlations between mortality risks of consecutive children and birth intervals. Equations driving the processes of infant mortality, birth spacing, and fertility are estimated jointly to account for the various sources of endogeneity. Each of the equations incorporates a mother specific unobserved heterogeneity term. The estimates of the causal parameters therefore remain consistent in the presence of reverse causality or unobserved heterogeneity. The model has an equation for mortality (neonatal mortality in Bhalotra and van Soest; infant mortality in our study), for the birth interval, and for the decision to have another birth. Mortality depends on, among other things, the length of the preceding birth interval (for birth orders higher than one), age of the mother, gender of the child, socio-economic status of the family, religion, and an unobserved mother specific effect. The decision whether to have another child or not and the birth interval after a given birth until the next birth in turn depend on gender and survival status of previously born children, age of the mother, socio-economic status, religion, and unobserved mother-specific effects. The three mother-specific unobserved effects are allowed to be correlated to capture the possibility of common unobserved factors driving the various processes. The model is estimated with maximum likelihood, accounting for all the correlations and for censoring in the birth spacing equation (fertility may be incomplete at the end of the observation window).

In contrast to Bhalotra and van Soest (2008), who uses retrospective data from the Indian Demographic and Health Survey, we use prospective data from the Demographic and Health Surveillance System, Matlab, Bangladesh, following mothers residing in the study area over time. This has the advantage that several covariates, such as indicators of socio-economic status and environmental factors such as availability of drinking water) are observed at the relevant points in time when children are born (rather than at survey time in the retrospective data). Moreover, it avoids recall error in, for example, the dates when children were born. A second feature that is specific to our data is that the study area is randomly split into villages with standard government provided health services: the comparison area and villages with additional extensive health services, such as more health clinics and regular visits of health officers: the ICDDR,B area or treatment area (Bhatia 1983; Van Ginneken et al. 1998). Comparing the model estimates for the two areas gives insight in how the extensive health services influence affect birth spacing, mortality, and fertility and the various relations between these processes.

3.2 Overview of causal mechanisms

According to the classical demographic transition theory, child mortality affects fertility in two ways: physiological/biological changes and behavioral/replacement effects. The physiological effect can be explained by the fact that breastfeeding is interrupted with a child death, and consequently, the postpartum infecundable period is shortened (Van Ginneken 1974; Mondot-Bernard 1977; Knodel 1978). As a result under ineffective use or non-use of contraception, the mother is able to conceive the next child sooner, leading to a shorter birth interval and, possibly, higher fertility. The effect of child mortality on birth intervals is closely linked to the influence

of breastfeeding on fertility (Knodel 1978). With reference to the behavioral changes, the association between the death of a child and the risk of child- bearing has been attributed to two main strategies of reproductive behavior: replacement and hoarding (Ben-Porath 1976; Wolpin 1998). Hoarding refers to the fertility response to *expected* mortality of offspring and replacement is the response to the *experience* of child death. These behaviors are closely related to the total number of surviving children that parents wish to have when they get older. Hoarding and replacement are substitutes in the sense that if families expect high mortality and respond to it by hoarding, fertility should not respond as strongly to actual mortality.

A shorter gap between births, in turn, seems to increase the mortality risk of the next child, particularly in the neonatal and post-neonatal period, because the mother has not recuperated physiologically from the previous birth (DaVanzo and Pebley 1993; Scrimshaw 1996). Hence vulnerable families can be caught in a death-trap: an endogenous process that creates persistence in death risk within families. The causal mechanisms make this a case of genuine state dependence, in the sense that the death of a child causes an increase in the risk of death of the subsequent sibling in the family (Arulampalam and Bhalotra 2006). Another reason for this might be sibling competition: the effect of diminishing sources of food and care per head as the number of dependent members of a family increases (Cleland and Sathar 1984). A further possibility is that a child death leaves the mother depressed. This may affect the mother's behavior and attitude of preventing health problems in relation to the next child. As a result, the health of her subsequent child is also compromised, both in the womb and in early infancy (Steer et al. 1992; Rahman et al. 2004).

Other causal mechanisms apart from birth-spacing may also play a role in explaining death-clustering, both negatively and positively. Learning effects may lead to a negative effect of child death on the death risk of the next child. For example, if the older sibling died because of diarrhea or acute respiratory illness (ARI) – two leading causes of child death in developing countries, explaining almost half of all deaths in Bangladesh (NIPORT et al. 2005) - the mother will often want to learn how to prevent a death caused by diarrhea or ARI. Another mechanism leading to negative state-dependence effect is competition for family resources. If the previous child is dead, the next child competes with fewer siblings, potentially improving its survival chances (Alam and David 1998).

There is evidence to suggest that son preference exists in societies like Bangladesh, where there are strong patrilineal family systems (Chowdhury et al. 1976). It is therefore likely that a couple may want to have another child soon after the death of a son until the desired number of sons is achieved. One study in Bangladesh shows that the median birth interval is shorter when the dead child was a boy or when it was survived by fewer than two brothers (Sufian and Johnson 1989). Another study using data on Ghana DHS shows that the probability of progression to the next child is about 32% higher if a male child dies than if a female child dies (Nyarko et al. 2003).

Observed clustering in infant or child mortality of successive children may be due to birth interval effects but also to some other factors. Most studies of birth spacing and childhood mortality do not appropriately control for whether the previous child died or survived as well as unobserved heterogeneity. Exceptions are Arulampalam and Bhalotra (2006) in India and Omariba et al. (2008) in Kenya. Findings from these studies reveal that the causal effect of previous mortality is overestimated when unobserved heterogeneity is not accounted for.

3.3 Literature review

Several researchers have investigated the relationship between child mortality and fertility, especially in developing countries, and have demonstrated firm evidence of an increased probability of pregnancy among women who lost their child (for example, Harrington 1971; Chowdhury et al. 1976; Ben-Porath 1976; Park et al. 1998). In Bangladesh, Chowdhury et al. find that infant death shortened median birth interval from 37.2 to 24.1 months. A study in Ghana shows that the death of the previous child shortens the length of interval by 11 months and confirms that the parity progression ratio is higher if an infant dies than if it survives (Nyarko et al. 2003). Ben-Porath (1976) studied Jewish women born in Asia or Africa and a group of women born in Europe or America or Israel and found significant replacement effects. Bhalotra and van Soest (2008) suggest that for every neonatal death in India, 0.37 extra children are born.

On the other hand, studies investigating effects of birth-spacing on mortality have observed a close association between a short preceding birth-interval and mortality (Zenger 1993; Guo 1993; Miller et al. 1992; Koenig et al. 1990; Alam and David 1998), and this association has been observed to be stronger when the preceding sibling survived compared to when it died (Zenger 1993; Koenig et al. 1990; Alam and David 1998). This may imply that the birth interval effect on mortality is confounded by the association between the mortality risks of successive births. In Bangladesh Demographic and Health Survey 2007 (NIPORT et al. 2008) the percentage of births that occur within a very short interval (less than 18 months) is six times higher for children whose prior sibling died than for those whose prior sibling survived. Multivariate analyses with large datasets from a number of different settings suggest that birth-intervals of at least three years are associated with better health outcomes for children (Conde-Agudelo et al. 2006; DaVanzo et al. 2004; Rutstein 2005).

Based on Matlab data, DaVanzo et al. (2008) report how detrimental effects of short birth intervals vary by pregnancy outcomes. For mortality in the first week of a child's life, they find that birth interval effects are greater if the sibling born at the beginning of the birth interval died than if it survived. After one month, however, the effects are greater if the previously born sibling was still alive. Their findings are in the line with the maternal depletion hypothesis and with sibling competition. However, in their analysis they did not account for endogeneity in the birth spacing or for correlated risks of death among different births of the same mother. In their

conclusions they emphasized that this remains a significant area for future research using Matlab data, and this is in essence the contribution of this paper.

3.4 Policy implications

Understanding the mechanisms of causality will help policymakers to determine how to allocate limited resources for effective interventions. For example, scarring effects can explain that the death of a previous child increases the death risk of the next child. In the patriarchal society of Bangladesh where son preference is very high (Chowdhury et al. 1990), it is expected that the scarring effects are higher if the deceased child was a boy and the index child is a daughter. If this appears to be the case, policymakers can pay more attention to a gender discrepancies campaign at the national level or can launch a female empowerment program, educating females up to a certain level. Another example can be derived from the relationship between birth-spacing and child mortality, especially neonatal or post-neonatal mortality, due to mother's depletion. If this is important, policy makers can focus on birth-intervals and start campaigning to promote a minimum birth-interval that will enable mothers to recuperate physiologically from child birth and prepare them for future births.

Furthermore, if we find that mother-specific effects are important, this may suggest that women who have experienced the loss of a child will be at higher risk of losing a second or third child. It can also help us to understand how a particular intervention (e.g. safe drinking-water) affects other components of health. For example, safe drinking-water was supplied to reduce childhood mortality but in reducing child mortality it also may help to lower the total fertility rate and hence may, in turn, also increase the birth-spacing between two births, giving more insight in how these particular mechanisms work.

3.5 Data

3.5.1 Study sample

Since 1966 ICDDR,B maintained a Health and Demographic Surveillance System (HDSS) in Matlab, aiming to support the Health and Family Planning programme in Bangladesh. Matlab is an area located in 60 km southeast of Dhaka in which all births, deaths, causes of deaths (through verbal autopsy), pregnancy histories, migrations in and out of the area, marriages, divorces, and several indicators of socioeconomic status are recorded for the complete population of about 220,000 people.[3] The HDSS data on the timing of pregnancy outcomes and deaths are thought to be of very high quality because they have been collected during regular visits (every two weeks until the late 1990s and every month since then) by well-trained female community health workers (CHWs; see, e.g., D'Souza 1981 or van Ginneken et al. 1998). We combined the health and demographic surveillance system data from 70 villages in the ICDDR,B area and 79 villages

[3] For details on surveillance system, see chapter 2 or Saha and van Soest (2011).

in the comparison area obtained from 1 July, 1982 until 31 December, 2005 (the study period). Data from before 1 July 1982 have not (yet) been made available for research.

The complete data set has records on about 63,000 mothers, with more than 165,000 child births – including live singleton births, multiple births, and still births. For our purposes, we selected a subsample of mothers without multiple births [4] and with complete [5] live birth information who were continuously living in the Matlab area since the birth of their first child. This implies that we deleted mothers who migrated out of Matlab during the period under study. Moreover, we discarded stillbirths. [6] Finally, we have excluded the children born in three villages which shifted from the ICDDR,B area to the comparison area in 2000. This leads to working samples of 31,968 children and 13,232 mothers in the ICDDR,B area and 32,366 children and 11,856 mothers in the comparison area.

3.5.2 Variables and descriptive statistics

Table 1 presents sample means (percentages of outcome 1 for dummy variables) by area. In the ICDDR,B area, 5.09 percent of all live births resulted in infant death; 10.66 percent of all families experienced at least one infant death and 0.79 percent lost all their children in infancy. The percent of infant death among first born is 6.70, substantially higher than among children of higher birth order (3.95 percent). In the comparison area, infant death was more common: 6.82 percent of all children where 8.90 percent among first born; 5.62 percent among higher birth orders. Of all families, 15.66 per cent experienced at least one infant death and 1.08 per cent lost all their children. About 20.6% birth intervals are shorter than or equal to 24 months in the comparison area, compared to about 12.9 % in the ICDDR,B area.

The average number of children born per mother is 2.42 in the ICDDR,B area and 2.73 in the comparison area; 19 percent of families had more than three children in the ICDDR,B area, compared to 29 percent in the comparison area (not reported in the table). No differences between areas are observed in the mother's average age at birth. Mothers have a somewhat lower education level in the comparison area than in the ICDDR,B area, on average. In the comparison area, mothers less often have access to the more hygienic source of drinking water (tubewell/filter) and live much farther away from the nearest health facility (7.1 kilometres on average, compared to 1.9 kilometres in the ICDDR,B area).

Figure 1, based upon non-parametric regressions of infant mortality on the preceding birth interval, depicts a sharp decline in infant mortality rates when birth intervals increase in both ICDDR,B and comparison area. At short birth intervals, the probability of infant death is

[4] We eliminated multiple births as children of a multiple birth face much higher odds of dying requiring a separate analysis, as has been documented in the demographic literature.

[5] To have a mother's complete birth information during the study period we have calculated parity (total number of live births) from the pregnancy history variables. We have checked parity and birth dates of all children. For example, if a mother has parity four, this means this mother has had four live births, so the birth dates of four children should be available in the file and this mother will appear four times as giving birth. If this was not the case (e.g., if a child was born outside of the Matlab area or before study period or deleting multiple births may caused incomplete birth information of a mother), we have deleted all children's records of this mother.

[6] One reason why we eliminated stillbirths is that gender, an important covariate in our analysis, is missing for stillbirths. We define birth intervals as intervals between reported dates of live births, ignoring stillbirths in between live births

highest, and it falls with birth interval length until an interval length of about 4.5 years (exp(4)=54 months). Particularly in the ICDDR,B area, the survival chances are quite stable and even seem to increase a little when birth intervals increase beyond 4.5 years. This pattern is in line with the wide demographic literature on this issue; see, for example, Figure 1 in Bhalotra and van Soest (2008).

Figures 2 and 3 show the distributions of the log birth interval by survival status of the previous child and gender in the ICDDR,B area and the comparison area. In both areas, there is a large difference in the distributions of the birth interval after infant death and after infant survival. This difference is much larger than the difference in the Indian state Uttar Pradesh found by Bhalotra and van Soest (2008, Figure 2). In the ICDDR,B area, the median birth intervals are 20 months after an infant death and 48 months otherwise (averages are 23 and 51 months). They are 17 and 37 months (averages are 22 and 42 months) in the comparison area. However, no significant difference is observed in these distributions by survival status and gender.

3.6 Model Specification

In this section we present the econometric model. This is similar to the model in Bhalotra and van Soest (2008; see also their online appendix for details), but we do not incorporate local community effects (although this would be a straightforward extension, in principle; as a consequence, our standard errors may be somewhat underestimated).

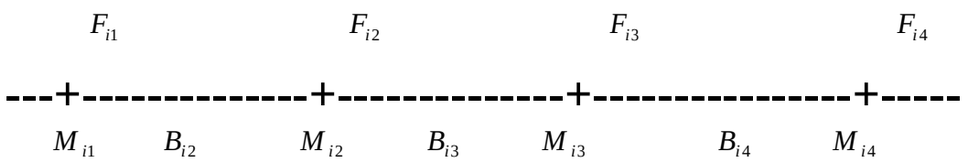
The endogenous variables in the model are the following, with *i* denoting a mother and *t=1,..,*T_i denoting her consecutive live births:

M_{it}: Infant mortality dummy: 1 if child *t* dies; 0 if it survives the first twelve months after birth.

F_{it}: Decision to have another child (1) or not (0).

B_{it}: Log birth interval preceding birth of child *t* (*t>1* only*)*

The sequence of events underlying the structure of the model, is illustrated in the following time line:



We do not explain the timing of the first birth. The first event we explain is infant survival of the first born child M_{i1}. The second is the decision to have more children ($F_{i1}=1$) or not ($F_{i1}=0$); this decision is never observed directly, but if a second birth is observed we know that $F_{i1}=1$. If not, this can be because $F_{i1}=0$ or because the next birth interval is too long in the sense that it exceeds the observation window or the woman's fertile age (set to 45 years).

If $F_{i1} = 1$ and if the birth interval is not too long, we observe the birth interval B_{i2}. The second born child can die during infancy or survive, etc.: the sequence of events continues until the mother decides not to have more children ($F_{iT} = 0$) or at the end of her fertile period (age 45) or the observation window (December 2005).

The model we use is recursive in the sense that each dependent variable may depend on outcomes realized earlier in the sequence of events, but not on future outcomes. Moreover, each outcome may depend on unobserved factors common to all children of a given mother, treated as unobserved individual (mother specific) effects. We use probit equations for the binary outcomes (infant mortality of each child; fertility decision after each birth) and a regression equation for the continuous outcomes (log birth intervals). Below we discuss the equations for the various outcomes in detail.

Infant mortality

The specification is similar to that in Bhalotra and van Soest (2008). For higher birth orders, a dynamic probit equation with (random) mother specific effects is used. The explanatory variables include the preceding birth intervals and variables related to the mother's age at birth, which is a function of previous birth intervals: For child t ($t=2,\ldots,T_i$) of mother i, the equation is

$$M_{it}^{*} = X_{it}\beta_m + Z_{it}\gamma_m + \alpha_{mi} + u_{mit} \qquad (1)$$

$$M_{it}=1 \text{ if } M_{it}^{*}>0 \text{ and } M_{it}=0 \text{ if } M_{it}^{*}\leq 0$$

Here X_{it} contains (functions of) the strictly exogenous variables, such as gender of the child, socio-economic status indicators of the household (mother's and father's education, etc.) and characteristics of the village where the household resides. Z_{it} is the vector of explanatory variables that are functions of previous outcomes (and are therefore not strictly exogenous), including the preceding log birth interval B_{it}, (functions of) age of the mother at birth t and, following the literature on scarring (see, for example, Arulampalam and Bhalotra 2006), survival status of the previous child M_{it-1}. The mother specific unobserved heterogeneity term α_{mi} captures unobservable time invariant characteristics influencing the infant mortality risk of all children in the family. The error term u_{mit} captures idiosyncratic health shocks specific to child t. We assume that the u_{mit} follow a standard normal distribution, independent of each other and of all covariates, and that α_{mi} is normally distributed with mean 0 and variance $\sigma^2_{\alpha m}$ independent of all u_{mit} and X_{it} (but not of Z_{it}).

For mortality of the first child, a separate equation is needed, since in this case there is neither a preceding birth interval nor a preceding mortality outcome. Age at first birth is assumed to be strictly exogenous (and included in X_{i1}). The equation for the first child's infant mortality is then given by:

$$M_{i1}^{*} = X_{i1}\beta^{1} + \theta\alpha_{mi} + u_{mi1} \qquad (2)$$

$M_{i1}=1$ *if* $M_{i1}^{*}>0$ and $M_{i1}=0$ *if* $M_{i1}^{*}\leq 0$

Here β^{1} and θ are (auxiliary) parameters to be estimated and the error term u_{mi1} is assumed to satisfy the same assumptions as the other u_{mit} .

Birth-spacing

For a mother who has given births to T_i children, we observe the exact log durations in between two consecutive births $b_{2i},\ldots,b_{T_i i}$ preceding births $2,.....,T_i$. We model these intervals using the following equation:

$$b_{it} = X_{it}\beta_b + Z_{it}^{b}\gamma_b + \alpha_{bi} + u_{bit} \qquad (3)$$

Here X_{it} denotes the vector of strictly explanatory variables, as before.[7] Z_{it}^{b} includes survival status of the preceding sibling and family composition variables (functions of the numbers of surviving girls and boys). The unobserved heterogeneity term α_{bi} captures unobserved time invariant characteristics of the mother (or her household or village) influencing the birth interval. The error term u_{bit} captures idiosyncratic errors. We assume that the u_{bit} follow a normal distribution, independent of each other and of all covariates, and that α_{bi} is normally distributed independent of all u_{bit} and X_{it} (but not of Z_{it}^{b}).

Fertility decisions and right censoring

There is right-censoring in the data since some mothers will not have completed their fertility at the time of the survey. After the end of the observation window (ultimo 2005), some mothers will still have another birth, and others will not. In principle, this could be captured by the model as it is described until now, with a birth interval after the last observed birth that lasts longer than till the end of 2005. Following Bhalotra and van Soest (2008), however, the model fit can be improved substantially by adding a separate equation reflecting the possible decisions to stop having children after each birth. This improves the fit since it can explain why some mothers who are still of reproductive age have no more births long before the end of the observation window. (We assume that women older than 45 years are no longer of reproductive age - an age beyond which very few births are observed in our data.) Without the additional equation, this would have to be explained by a very long birth interval.

The equation determining whether the woman continues to have children after birth *t* ($F_{it}=1$) or not ($F_{it}=0$) is specified as follows:

$$F_{it}^{*} = X_{it}\beta_f + Z_{it}^{f}\gamma_f + \alpha_{fi} + u_{fit} \qquad (4)$$

$F_{it} = 1$ if $F_{it}^{*}>0$ and $F_{it} = 0$ if $F_{it}^{*} \leq 0$

[7] Another determinant of birth spacing would be the use of contraceptives. We do not include this in X_{it} since it is not observed in the comparison area. Moreover, it may well be correlated with the unobservables in the model and therefore endogenous. Chapter 4 introduces an extension of the model of the current chapter where contraceptive use is modeled as an additional dependent variable. The current model can be seen as a semi-reduced form of this model. If, for example, infant mortality reduces the next birth interval because contraceptives are not used, then in the current model, this will be part of the effect of infant mortality on birth spacing.

As before, X_{it} denotes the vector of strictly exogenous explanatory variables. The vector Z_{it}^{f} includes survival status of the preceding sibling and family composition variables (based upon the number of surviving girls and boys). The mother specific unobserved heterogeneity term α_{fi} captures unobservable time invariant characteristics influencing the fertility decision after each child birth and the term u_{fit} captures idiosyncratic errors. We assume that the errors u_{fit} are standard normally distributed, independent of each other and of all covariates. The mother specific unobserved heterogeneity terms α_{fi} are normally distributed with mean 0 and variance $\sigma_{\alpha f}^{2}$, independent of all u_{fit} and X_{it}.

The outcome F_{it} is observed only partially. If birth t is not the last birth ($t<T_i$) then we know that the mother has decided not to stop having children, so that $F_{it} = 1$. But if $t=T_i$, she may have decided to stop having children ($F_{it} = 0$), but it may also be the case that the next birth interval extends beyond reproductive age or the end of the observation window ($F_{it} = 1$ and right censoring).

Confounding unobserved factors are controlled for by allowing arbitrary correlations amongst $\alpha_{fi,}$ α_{mi}, and α_{bi}. We will assume they are drawn from a three-dimensional normal distribution with zero mean and an arbitrary covariance matrix, independent of the X_{it} and of all the idiosyncratic error terms $u_{fit,}$ u_{mit}, and u_{bit}.

The equations of this model (equations (1)-(4)) are estimated jointly using simulated maximum likelihood. Conditional on the random mother specific effects, the likelihood contribution of a given mother can be written as a product of univariate normal probabilities and densities over all births following the order of observed events (see the time line) and accounting for the right censoring. The actual likelihood contribution is the expected value of the conditional likelihood contribution, with the expected value taken over the three unobserved mother specific random effects $\alpha_{fi,}$ α_{mi}, and α_{bi}. This three-dimensional integral is approximated using (smooth) simulated ML: Independent standard normal errors are drawn, and transformed into draws of the random effects using the parameters of the random effects distribution; the conditional likelihood contribution is then computed for each set of draws and the mean across R independent draws is taken. If $R\rightarrow\infty$ with the number of mothers N, this gives a consistent estimator; if draws are independent across households and $R\rightarrow\infty$ faster than $\sqrt{N}$, the estimator is asymptotically equivalent to exact ML (see, for example, Hajivassiliou and Ruud 1994). To reduce the sampling variance in the simulations, we used Halton draws (see Train 2003). The results we present are based on $R=100$ and we got very similar results for larger values of R.

We have checked the sensitivity of our parameter estimates for the number of the draws (comparing with different values of R) and the nature of the draws (using Halton draws with different seeds) and always got very similar results. This estimation procedure is quite similar to

the one used by Bhalotra and van Soest (2008); see also their online appendix for details and the likelihood contributions.

3.7 Estimation results

Mortality equation

The estimates of the mortality equation are given in Table 2.[8] Figures 4 and 5 are presented to help and interpret the parameters on lagged mortality, the log birth interval and its square, and the interaction of lagged mortality with the log birth interval. These figures sketch, for the two areas, the estimated mortality risk as a function of the birth interval separately for the cases where the previous child died and did not die during infancy, with other covariates set to their mean values. In the ICDDR,B area, the interaction term and lagged mortality are both significant. The significantly positive interaction term is in contrast with Bhalotra and van Soest's finding, but it is consistent with other studies (Conde-Agudelo et al. 2006; Whitworth and Stephenson 2002). For a given length of the birth interval, the "state dependence" effect of lagged mortality depends on the magnitude of the interval. For short birth intervals, state the mortality risk is larger if the previous sibling survived than if it died (negative state dependence), but for long birth intervals the difference changes sign (positive state dependence). This result is consistent with sibling competition for scarce family resources which are particularly needed when children are still very young. For long birth intervals, sibling competition plays much less of a role and other mechanisms such as depression due to the previous infant's death (cf. Bhalotra and van Soest 2008) may still matter. In the ICDDR,B area, for the case where the previous sibling did not die, the mortality risk falls with the birth interval until about the mean interval length and then flattens out. Surprisingly, a quite different pattern is found when the previous sibling died – in this case the mortality risk seems to increase with the birth interval length. Perhaps this is due to the relatively small number of observations and the rather small mortality risk in this case.

In the comparison area, the difference between the two curves is much smaller, and the mortality risk is consistently larger if the previous sibling survived than if it died (keeping the birth interval and other covariates and unobserved mother specific factors constant). This could reflect a learning effect but since the difference is statistically insignificant, we should probably not make too much of this. Both mortality risks in the comparison area are essentially falling with birth interval length, flattening out only after more than 50 months, much beyond the median birth interval length.

The estimated coefficients on the other covariates are in line with those in chapter 2 or Saha and van Soest (2011).The mother's age at birth has a significantly U-shaped effect in the ICDDR,B area with a minimum at about 30 years of age, whereas it is insignificant in the comparison area. Mortality risk is also U-shaped in birth order, but this is significant in the

[8] Results of the equation for mortality of the first child are not given in this chapter; they are very similar to those reported in chapter 2 or Saha and van Soest (2011).

comparison area only. The gender of the child is insignificant in both areas, implying that any forms of possible son preference have no notable effect on the infant mortality risk.

Mother's schooling is insignificant once the father's schooling is controlled for (it is significant for the mortality risk of the first born child – see chapter 2 or Saha and van Soest 2011). On the other hand, secondary schooling of the father significantly reduces infant mortality of higher birth orders in both areas. The dummy indicating whether the father is a day labourer, an index of lower occupational and socio-economic status, has a significant positive effect on mortality in both areas. The distance to the nearest health facility has a significant positive effect on infant mortality in the comparison area for higher order births, and the effect is even stronger for the first born child. The fact that distance plays no significant role in the ICDDR,B area is probably due to the fact that almost all families live rather close to a health facility in that area (see chapter 2 or Saha and van Soest 2011).

Those who used tube well or pipe water as a source of drinking water are less likely to see their children die in infancy, but this finding is significant for higher birth orders in the ICDDR,B area only. Over the various birth cohorts (the reference mother is born before 1966), mortality decreases sharply in the comparison area, while in the ICDDR,B area, the decreasing trend seems to level off for the younger cohorts.

Birth interval equation

Table 3 reports the estimates of the birth spacing equation. The dependent variable is log birth interval and thus the interpretation of the parameter estimates is in terms of percentage changes in the expected length of the birth interval. Death at infancy of the previous child shortens the subsequent birth interval by about 49% (exp(-0.6741)-1) in the ICDDR,B area and by about 46% in comparison area, consistent with the replacement hypothesis and existing findings (e.g. Chowdhury et al. 1976; Bhalotra and van Soest 2008). The size of the effect is much larger than in Bhalotra and van Soest (2008). The effects of the surviving boys and girls variables are consistent with son preference: In both areas, having at least one boy has a stronger positive effect on the birth interval than having a girl. The same applies to each additional boy. For example, in the ICDDR,B area, the ceteris paribus difference between the next birth interval of families with one boy and one girl is 6.5% (exp(0.1726-0.1099)). Comparing families with two boys and one girl and with one boy and two girls, it is 6.7% (exp(0.0978-0.0325)).

Birth intervals shorten with birth order (see for example, Miller et al., 1992). They are longer for the younger birth cohorts of mothers, which may explain part of the reduction in fertility over time. Mothers educated up to primary level have significantly longer intervals than mothers with no education, and this positive effect of education is even more pronounced for mothers with some secondary level education. In the ICDDR,B area, birth-spacing is hump-shaped in maternal age at previous birth with a maximum at about 35 years. In the comparison area, birth interval length essentially increases with the mother's age at the previous birth over the whole reproductive age range. This result is in line with the negative effect of maternal age

on the hazard rate of another conception found by Rahman and DaVanzo (1993, Table 2). Mothers in more developed villages with drinking water from a tube well or pipe water tend to have longer birth intervals.

Fertility equation

Table 4 presents estimates of the probability of having another child after each birth. In both areas, the most important variables in this equation are the family composition variables. Having at least one son or at least one daughter substantially and significantly reduce the probability to have further children, and the size of the effect is much larger in the comparison area than in the ICDDR,B area. There is no son preference in this respect in either area. On the other hand, if we consider the number of sons and daughters given there is at least one of each, we do find evidence of son preference: Each additional son substantially reduces the desire to have more children substantially (though less than the first girl), but additional girls have a much smaller effect (significant in the comparison area; small, insignificant, and of the wrong sign in the ICDDR,B area.

Fertility falls with the level of education of both mother and father, with mother's education having a larger effect. In both areas, Muslim families show a higher tendency to continue fertility than their non-Muslim counterparts. The desire for continued fertility falls with birth order in both areas (keeping family composition and other variables constant). Younger mothers at birth are less likely to continue fertility than older mothers. There are strong cohort differences in the comparison area where the younger cohorts less often want more children, but these cohort effects do not exist in the ICDDR,B area. Mothers in villages with access to tube well or pipe water as a source of drinking water are less likely to continue their fertility. In the comparison area, we also find that families living farther away from a health centre have a larger probability to have another child. These results are in line with a negative relation between socio-economic status and fertility. This is not the case for the result on the father's occupational status: in both areas we find that day labourers have smaller chances to have more children.

Unobserved heterogeneity

The estimates of the covariance matrix of the three unobserved heterogeneity terms are given in Table 5. The heterogeneity terms in all three equations are statistically significant but smaller than the idiosyncratic errors. The mother specific heterogeneity terms in the mortality equation explain about 23% (0.3014/(1+0.3014)) in the ICDDR,B area and about 6% (0.0625/(1+0.0625)) in the comparison area of the total unsystematic variation in infant mortality. For the birth spacing equation the idiosyncratic noise terms have estimated standard deviation 0.442 in the ICDDR,B area and 0.436 in the comparison area, and the unobserved heterogeneity terms explain 8.1% of the unsystematic variation in birth intervals in the ICDDR,B area. The small correlations between unobserved heterogeneity in the mortality and birth interval equations suggest that hoarding does not play much of a role in either area – hoarding would predict that women who know their children have high mortality risk tend to have shorter birth intervals in

order to be able to achieve their desired family size even if some children die, and to the extent this is not captured by observed covariates, this would imply a negative correlation between α_{mi}, and α_{bi}.

The heterogeneity terms in the fertility equation explain about 70% (comparison area) and 44% (ICDDR,B area) of total unsystematic variation. In both areas, a large negative correlation is observed between unobserved heterogeneity in both the birth interval and fertility equations, suggesting that mothers who desire many children also tend to use shorter birth intervals. This is consistent with the target fertility model of Wolpin (1978) and also in line with the finding of Bhalotra and van Soest (2008). The correlation between the individual effects in the mortality equation and the fertility equation is positive but not significant in the ICDDR,B area (the point estimate is 0.386 but rather imprecise), but significantly negative in the comparison area. We do not have a good explanation for this.

3.8 Simulations

To illustrate the importance of the various causal mechanisms between birth spacing, fertility, and infant mortality, we performed some simulations, in a similar way as the simulations in Bhalotra and van Soest (2008, Table 3). These simulations illustrate the main feature of our joint model: the fact that it incorporates various mechanisms that lead to associations between planning, birth spacing, fertility, and mortality outcomes, accounting for the effects of endogeneity in the timing of births (and therefore also age at birth etc.), birth intervals, and mortality risks.

The simulations use the covariates (including, for example, date of first birth) as observed for each mother in the actual sample. We then generated, for each mother in the sample, unobserved heterogeneity terms, error terms, and new outcomes (the dependent variables in our model) using the estimated parameters of each equation. The outcomes were generated recursively, using the timing of the events as sketched in Section 3.6. For example, for a given mother, we take the date of first birth as given and first generate the mortality outcome of the first child (using equation (2)). Given simulated mortality, we then generated the fertility decision after the first birth (equation (4)). If the fertility decision is positive, we then generate a birth interval, and update calendar time and age of the mother at her second birth. Given these variables, other covariates, and the previous mortality outcome, we then generate the mortality outcome of the second born child, etc. In this way we generate complete birth spacing, mortality, and fertility patterns for all mothers in the sample. To reduce simulation variance, this is repeated 25 times for each mother.[9]

Table 6 shows the results of some simulations based upon the estimated models for the two areas. It is important to note that we have checked the simulated means with the actual

[9] The simulations take the parameter estimates as given. In principle, it would be possible to compute a standard error for each simulation outcome by repeating the simulations for other draws of the model parameters from their estimated (asymptotic) distribution but this would require a substantial computational effort.

means and found very close each other. They illustrate the estimated importance of some of the mechanisms incorporated in the model, such as the effects of mortality on birth intervals and fertility, and the importance of state dependence and hoarding, similar as in Table 3 in Bhalotra and van Soest (2008). Column 1 summarizes the outcomes according to the benchmark simulation where all mechanisms that are incorporated in the model are active. As expected (unless the model would fit the data quite poorly), this column reproduces some features of the raw data, such as the differentials in infant mortality rates and median birth intervals between the two areas.

The other columns present percentage deviations from the benchmark for scenarios in which some behavioural or non-behavioural mechanisms are "switched off." Column 2 switches off the replacement effects of infant mortality on both birth intervals and the probability of having another child: while the estimates show that families respond to infant mortality by shortening the next birth intervals and increasing the number of births, this simulation produces the counterfactual outcomes that would arise if families would space their births and plan the number of births as if every child survived its infancy. The results in column 2 show that this increases median birth interval length by 5.9% and 6.3% in the two areas, consistent with other studies (see for example Chowdhury et al. 1976, pp. 259; Bhalotra and van Soest 2008, p.286). In other words, the replacement effect on the birth intervals reduces birth interval lengths by 5.9% and 6.3% in the two areas.

The replacement effect on the total number of births is larger in comparison area than in the ICDDR,B area (2.18%), mainly because the response of fertility decisions to the family composition variables is larger in the comparison area (Table 4). In the comparison area, the total replacement effect as a result of the infant mortality rate of 68.5 per 1000 live births is an increase in the number of births by 3.72% (37.2 births per 1000 births) that is, 0.54 births for every infant that died. In the ICDDR,B area, it is 2.18/5.18=0.42 births for every infant that died. Because of the longer birth intervals and the reduction in fertility, eliminating the replacement effects also has an indirect effect on infant mortality: it falls by 0.3% (0.14 per 1000 live births) in the ICDDR,B and by 3.6% (2.45 per 1000 live births) in the comparison area. In other words, replacement effects are responsible for a negligible fraction of all infant deaths in the ICDDR,B area and for a somewhat larger fraction of infant deaths in the comparison area.

Column 3 shows what happens if the direct effect of mortality of the previous child on survival chances is eliminated. (It does not eliminate replacement effects.) Since this direct effect was negative in both areas, eliminating it increases infant mortality: by 4.85% (2.51 infant deaths per 1000 live births) in the ICDDR,B area and by 1.56% (1.07 per 1000 live births) in the comparison area, in line with the larger state dependence effect in the ICDDR,B area. As discussed in the previous section, learning effects can explain this negative state dependence mechanism: if a mother experiences a child death due to diarrhoea or ARI then this mother often may want to learn how to prevent the death of her next child caused by diarrhoea or ARI.

Sibling's competition is another phenomenon that also explains negative state dependence because if the previous child is dead, the next child competes with fewer siblings, potentially improving its survival chances. Because of replacement behaviour, the larger infant mortality rates indirectly also shorten birth intervals and increase total fertility. In the ICDDR,B area, but these indirect effects are very small.

Hoarding implies that families respond to expected child mortality by adjusting birth spacing and family planning behaviour. In our model this leads to a correlation between unobserved heterogeneity terms in the mortality equation on the one hand and the birth spacing and fertility equations on the other hand. In the simulation presented in column 4, we eliminate these correlations, taking out the part of mother specific unobserved heterogeneity in birth intervals and fertility decisions that is correlated with the unobserved heterogeneity term in the mortality equation (so that the variance of the unobserved heterogeneity terms α_{fi}, and α_{bi} is also reduced). Since this does not change the *average* values of α_{mi}, and α_{bi}, the direct effects on birth intervals and total fertility are very small. In the ICDDR,B area, the estimated correlation between α_{fi}, and α_{mi} was positive, implying that mothers with high risk births tend to have higher fertility. Eliminating this correlation therefore reduces the number of high risk births (and increases the number of low risk births), so that infant mortality falls. Column 4 shows that the estimated reduction is 2.26%, or 0.18 infant deaths per 1000 live births. In the comparison area, the estimated covariance structure is very different (with a negative correlation between α_{mi} and α_{fi}) and the effect on mortality has the opposite sign. The increased infant mortality rate also leads to a modest increase in total fertility, due to replacement (cf. column 2).

The final simulation (column 5) illustrates the importance of son preference in family planning. We suppress son preference by simulating counterfactual birth spacing and fertility decisions assuming that families behave as if all their children were boys. This would lengthen the median birth interval by 3.9% in the ICDDR,B area and by 3.1% in the comparison area, and it would reduce total fertility by 3.4% in the ICDDR,B area and by 5.7% in the comparison area. Although these behavioural changes would reduce the infant mortality rates for higher order births, the ultimate effect on the infant mortality rate is positive. This is due to a composition effect: since the number of higher order births is reduced, the weight of relatively risky first births in the total infant mortality rate is increased.

3.9 Discussion and conclusion

We jointly analyze infant mortality, birth spacing, and total fertility of children in a rural area in Bangladesh, using longitudinal data from the Health and Demographic Surveillance System (HDSS) in Matlab, Bangladesh. While Bangladesh has experienced a sharp decline in under-five mortality and total fertility during the past decades, further reducing both child mortality and total fertility remains an important concern, with the aim to achieve the United Nations Millennium Development Goals 4 and 5 (UNDP 2003). To distinguish causal mechanisms from unobserved heterogeneity and reverse causality, we use dynamic panel data

techniques, building on recent work by Bhalotra and van Soest (2008). We compare the pattern of the results in a treatment area with extensive health services and a comparison area with the standard health services provided by the government.

Controlling for birth spacing, unobserved heterogeneity, and a large set of socio-economic and cultural covariates, we find negative state dependence in both areas and this relationship is significant in the ICDDR,B area. This finding is unique among studies of infant mortality. For example, in Matlab, Bangladesh, Alam and David (1998) find higher risks in sibling's death if previous sibling died at same age (either neonatal or post-neonatal period). DaVanzo et al. (2008) using the similar data sets find higher mortality risks among siblings in the post-neonatal period, though they also find negative state dependence in the neonatal period (explained by the sibling competition hypothesis). In India, Arulampalam and Bhalotra find that, keeping other factors constant, infant death of the previous sibling increases the likelihood of infant death by between 2.2 percent points (West Bengal and Punjab; two of the richest states) and 9.2 percent points (Haryana). In Kenya, Omariba et al. (2008) find a positive scarring effect of 4.8 percent points. These studies do not control for birth intervals. In chapter 2 or Saha and van Soest (2011, Table 5), we also found negative state dependence when keeping preceding birth intervals constant, but the negative effect is about two to three times larger in the current study, which emphasize the importance of allowing for the endogeneity of birth-spacing in the model.

Even though they have shorter birth intervals and higher fertility, Muslims exhibit lower mortality in both ICDDR,B and comparison area, similar to what is found for India (Bhalotra et al., 2010a,b). It suggests that there may be something different about women who have higher mortality but lower fertility for example desired fertility. If this analysis could be restricted to birth order two then probably we would able to see a relationship (as expected) between mortality, fertility and religion. Cultural beliefs and practice might be a leading cause of such higher mortality risks among Hindus because Hindu women in rural Bangladesh during their births live in a poorly constructed (mainly thatches) house, and are not given warm clothes for baby and mother (personal observation).

Our simulation results enhance learning effects in ICDDR,B area, and this finding supports Ben-Porath's view that learning process at work in the sequential framework when experiencing mortality affects expected mortality. Ignoring the direct effect of lagged mortality on mortality of next child (learning effects/sibling competition) increases mortality by 4.9% in ICDDR,B area and by 1.6% in comparison area. We also find evidence of causal effects in both directions: a short preceding birth interval reduces survival chances of infants, and an infant death shortens the time until the next birth (replacement behaviour), and also increases the probability of next birth. As a result of replacement 0.54 births of all births are born for every infant death (and 0.51 births survive the first 12 months) in the comparison area whereas in the ICDDR,B area 0.42 children are replaced for every infant death.

There is no evidence of hoarding in the sense that frailty would be negatively correlated with fecundity (see small correlation in Table 6). This finding indicates that there is no evidence that a mother who knows that her child is at relatively large risk of infant death anticipates this by reducing the length of next birth interval. Similar finding is observed in other study in India (Bhalotra and van Soest, 2008).

In both areas, higher mortality risks are observed after long birth intervals after an infant death, which suggests that after an infant death and a long interval the mother may behave (both physiologically and behaviorally) as a first born mother (see Conde-Agudelo et al. 2006). This risk is large in ICDDR,B area. It might be the case also that long intervals disproportionately affect some mothers who are at the end of their reproductive span; analyzing this is left for future research. Furthermore, the finding in ICDDR,B revealed reduced mortality risks at shorter birth intervals while a mother experienced previous infant death and this finding contrasts other findings (for example, DaVanzo et al. 2008). The current study reveals the birth intervals minimizing the mortality risk are about 50 months in ICDDR,B area and 60 months in the comparison area for the majority of cases where the previous child did not die during infancy.

Estimates of fertility behaviour are consistent with son-preference where having more surviving boys significantly reduces the probability of having a next child and this effect is strong in the comparison area. This finding is revealed comparing the coefficients of lagged mortality and number of surviving boys and girls in the fertility equation. According to literature we expect son preference in a population that has access to contraception and higher levels of contraceptive use (see Rahman and DaVanzo 1993).

Those who used tube well or pipe water as a source of drinking water are less likely to see their children die in infancy, and in turn decreases fertility and increases birth spacing, reflects social multiplier effects and an advantage of simultaneous modeling approach. This finding is unique in this study and guidance for policies to enhance safe drinking water. We find evidence that mortality risks altered with reproductive behaviour and by socio-economic indicators for example age at birth, birth-spacing and maternal education, they have implications for the advice that should given women about pregnancy spacing, age at birth. Indeed, this advice is more important for the women with low socio-economic status (e.g., illiterate couple, and less access to hygienic environment).

Concerning policies targeted at achieving the fourth millennium development goal to reduce under-five mortality, our findings highlight the important role of extensive maternal and child health interventions: comprehensive health infrastructure, providing extensive health services and health information in the ICDDR,B area, strengthens learning effects that can reduce mortality risk in the ICDDR,B area. Indeed, it is worthwhile to mention that the effect of interventions in the ICDDR,B area may have spilled over into the comparison area over the period (LeGrand and Phillips, 1996). Thus, it is important to conduct a similar analysis in other

areas of Bangladesh where perhaps we can expect positive scarring. In line with our companion paper (Saha and van Soest 2011), the finding in this paper also suggests (large unobserved heterogeneity in ICDDR,B area) for implementing policies that increase equity in interventions may help to further reduction in infant mortality.

The main goal of this study is to explore the causal mechanisms between infant deaths and total fertility, and how birth spacing shapes this relationship. We compared the pattern of this relationship between two areas and found several significant different differences, suggesting that one model for both areas would be too restrictive.[10] In a decomposition analysis in chapter 2 (Annex), we found that the nearest health facilities contributes substantially to explaining the existing differences in infant mortality between the two areas, suggesting that the extensive maternal and health interventions in the ICDDR,B area help to explain these differences (also see Hale et al., 2006). We also tried using dummies for whether specific interventions were introduced at the time of birth, but these were not significant.

[10] Of course we could also combine the two areas and allow for interactions where necessary (according to tests). In chapter 2 we performed such a joint analysis but we found hardly any efficiency gain. Since the interactions also do not help to make interpretation easier, we did not pursue this here.

Tables

Table 1. Descriptive statistics, Matlab, 1982-2005.

Variables	ICDDR,B area	Comparison area
Infant deaths (all live-births) (%)	5.09	6.82
Infant deaths excluding first-borns (%)	3.95	5.62
Infant deaths among first borns (%)	6.70	8.90
Families with no infant deaths (%)	89.34	84.34
Families in which all births die in infancy (%)	0.79	1.08
Preceding birth interval in months (%)		
<=24 months	12.93	20.65
25-36 months	19.92	32.73
>=37 months	67.14	46.63
Age of mother at first birth*	21.16 (3.23)	21.08 (3.21)
Age of mother at birth*	24.70 (5.03)	24.58 (4.85)
Mother's education level (%):		
No education	48.48	50.50
Some primary education	24.86	25.51
At least some secondary education	26.66	23.99
Mother Muslim (%)	82.71	89.85
Child male (%)	50.97	51.12
Birth order (%)		
1	41.39	36.63
2	28.93	26.74
3	17.62	18.26
4+	12.06	18.36
Father's education level (%):		
No education	55.67	56.28
Some primary education	22.65	24.15
At least some secondary education	21.68	19.57
Father day labourer (%)	19.61	20.96
Drinking water tubewell/piped water (%)	87.76	76.91
Distance to health facility (km) *	1.87 (0.98)	7.07(4.04)
Number of mothers in sample	13,232	11,856
Number of children in sample	31,968	32,366

*: Means and standard deviations (in parentheses).

Table 2. Estimation Results Mortality Equation, Birth Order > 1 (Equation (1)).

Covariates	ICDDR,B area		Comparison area	
	estimates	s.e	estimates	s.e
Previous sibling died	-1.9904**	0.4637	-0.2703	0.3712
Preceding birth interval (log)	-2.7871**	0.4772	-1.7239**	0.4191
Preceding birth interval square (log)	0.3565**	0.0644	0.2094**	0.0571
Log birth interval_lagged mortality	0.5471**	0.1384	0.0648	0.1157
Male	0.0352	0.0399	0.0111	0.0309
Muslim	-0.0275	0.0604	-0.0503	0.0516
Birth order	0.0494	0.1091	-0.1512*	0.0583
Birth order square	-0.01327	0.0152	0.0199*	0.0069
Mother's birth cohort:				
1966-1970	-0.0213	0.0548	-0.1516**	0.0400
1971-1975	-0.1513*	0.0674	-0.3055**	0.0486
1976+	-0.1878*	0.0807	-0.5461**	0.0619
Mother's age at birth	-0.1260**	0.0371	-0.0321	0.0333
Mother's age at birth square	0.0020**	0.0006	0.0004	0.0006
Mother's education some primary	-0.0616	0.0537	0.0096	0.0400
Mother's education at least some secondary	-0.2305**	0.0697	0.0896	0.0543
Father's education some primary	0.0604	0.0506	0.0286	0.0393
Father's education at least some secondary	-0.2305**	0.0684	0.1312*	0.0500
Father's occupation is day labourer	0.1271*	0.0636	0.1239*	0.0452
Source of drinking water: tubewell /piped	-0.1767*	0.0633	0.0194	0.0395
Distance to health facility (km)	-0.0002	0.0227	0.0064	0.0039
Constant	5.4656**	0.9774	2.8594**	0.8554

* t-value<3; ** t-value>3

Notes:

Reference categories of categorical variables used in the model: female, non-Muslim, no schooling years, no access to piped water, not day labourer, mother born before 1966.

Table 3. Estimation Results Log Birth Interval Equation, Birth Order > 1 (Equation (3)).

Covariates	ICDDR,B area		Comparison area	
	estimates	**s.e**	**estimates**	**s.e**
Previous sibling died	-0.6741**	0.0178	-0.6107**	0.0147
First boy surviving	0.1726**	0.0203	0.1226**	0.0160
First girl surviving	0.1099**	0.0198	0.0723**	0.0161
After first boy, number of boys surviving	0.0978**	0.0191	0.0764**	0.0143
After first girl, number of girls surviving	0.0325	0.0186	0.0197	0.0136
Male	-0.0104	0.0103	-0.0306	0.0092
Muslim	-0.0145	0.0105	0.0090	0.0111
Birth order	0.1136**	0.0219	0.0746**	0.0160
Birth order square	-0.0228**	0.0026	-0.0136**	0.0018
Mother's birth cohort:				
1966-1970	0.0659**	0.0098	0.0461**	0.0090
1971-1975	0.1556**	0.0116	0.1072**	0.0109
1976+	0.2320**	0.0130	0.1554**	0.0131
Mother's age at birth	0.0262**	0.0065	0.0207*	0.0082
Mother's age at birth square	-0.0004*	0.0001	-0.0002	0.0002
Mother's education some primary	0.0372**	0.0091	0.0565**	0.0083
Mother's education at least some secondary	0.0035**	0.0107	0.1247**	0.0101
Father's education some primary	-0.0054	0.0088	-0.0171**	0.0081
Father's education at least some secondary	0.0372	0.0098	0.0066	0.0089
Father's occupation is day labourer	-0.0046	0.0121	-0.0440**	0.0104
Source of drinking water: tubewell /piped	0.0414**	0.0101	0.0243**	0.0080
Distance to health facility (km)	0.0042	0.0037	-0.0009	0.0008
Constant	3.0807**	0.0801	3.0370**	0.0982
Sigma error in birth interval equation	0.4422**	0.0029	0.4356**	0.0027

* t-value<3; ** t-value>3

Notes:

Reference categories of categorical variables used in the model: female, non-Muslim, no schooling years, no access to piped water, not day labourer, mother born before 1966.

Table 4. Estimation Results Decision to Have Next Child (Equation (4)).

Covariates	ICDDR,B area		Comparison area	
	estimates	s.e	estimates	s.e
Previous sibling died	-0.15572	0.1004	-0.2092*	0.0991
First boy surviving	-0.5969**	0.1568	-1.2778**	0.1699
First girl surviving	-0.5211**	0.1537	-1.2930**	0.1641
After first boy, number of boys surviving	-0.3367*	0.1443	-1.1801**	0.1503
After first girl, number of girls surviving	0.0307	0.1403	-0.6347**	0.1104
Male	-0.0462	0.0462	-0.0197	0.0485
Muslim	0.6076**	0.0787	0.3869**	0.1001
Birth order	-0.3857*	0.1640	0.3148**	0.1015
Birth order square	0.0100	0.0089	-0.0173*	0.0069
Mother's birth cohort:				
1966-1970	0.0418	0.0473	-0.1730*	0.0672
1971-1975	0.1051	0.0720	-0.5095**	0.0991
1976+	1.2814	0.6862	-0.9052**	0.1573
Mother's age at birth	0.0008	0.0333	-0.0613	-0.0613
Mother's age at birth square	-0.0026	0.0007	-0.0028**	-0.0028
Mother's education some primary	0.0331	0.0539	-0.1940*	0.0711
Mother's education at least some secondary	0.3843**	0.0790	-0.5045**	0.1017
Father's education some primary	0.0229	0.0522	0.1156	0.0664
Father's education at least some secondary	-0.1248*	0.0603	-0.0957	0.0770
Father's occupation is day labourer	-0.5451**	0.0790	-0.4155**	0.0862
Source of drinking water: tubewell /piped	-0.1205*	0.0554	-0.1453*	0.0606
Distance to health facility (km)	-0.0216	0.0181	0.0245**	0.0068
Constant	4.4781**	0.5415	6.9565**	0.9225

* t-value<3; ** t-value>3

Notes:

Reference categories of categorical variables used in the model: female, non-Muslim, no schooling years, no access to piped water, not day labourer, mother born before 1966.

Table 5. Mother specific unobserved heterogeneity.

	Mortality	Birth interval	Fertility
ICDDR,B area			
Covariance matrix			
Mortality	0.301**		
Birth interval	-0.012	0.017**	
Fertility	0.189	-0.099**	0.793**
Correlation matrix			
Mortality	1		
Birth interval	-0.167	1	
Fertility	0.386	-0.856**	1
Comparison area			
Covariance matrix			
Mortality	0.063**		
Birth interval	-0.0002	0.007**	
Fertility	-0.188**	-0.088**	2.306**
Correlation matrix			
Mortality	1		
Birth interval	-0.012	1	
Fertility	-0.495**	-0.698**	1

** t-value>3

Table 6. Simulations.

ICDDR,B area	**1**	**2**	**3**	**4**	**5**
Infant mortality	51.8/1000	-0.27	4.85	-2.26	1.63
Median birth interval	43.12	5.87	-0.20	0.70	3.87
Number of births (fertility)	2.43	-2.20	0.01	-0.20	-3.32
Number of survivors	2.31	-2.18	-0.26	-0.08	-3.40
Comparison area					
Infant mortality	68.5/1000	-3.57	1.560	2.208	0.43
Birth interval (months)	35.95	6.30	-0.20	-0.10	3.15
Number of births (fertility)	2.75	-3.72	-0.35	0.73	-5.68
Number of survivors	2.56	-3.47	-0.46	0.57	-5.71

Notes: Column 1 presents simulated outcomes for the benchmark model. Columns 2-5 show percentage deviations from the benchmark outcomes that arise when selected mechanisms are "switched off" as follows:

Column 2: no effect of infant mortality on birth interval or probability of having another child
Column 3: no direct effect of lagged mortality on mortality
Column 4: no correlation between unobserved heterogeneity in mortality equation and other equations (no hoarding)
Column 5: birth spacing and family planning as if all children are boys (no gender preference in birth intervals or probability of having another child)

Figures

Figure 1: Infant mortality and preceding birth interval

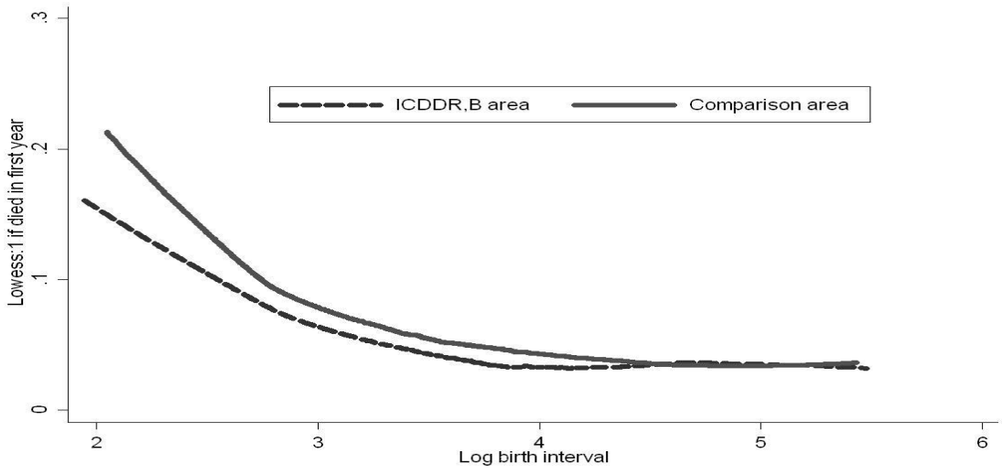


Figure 2: Birth intervals by survival status and gender of previous child, ICDDR,B area

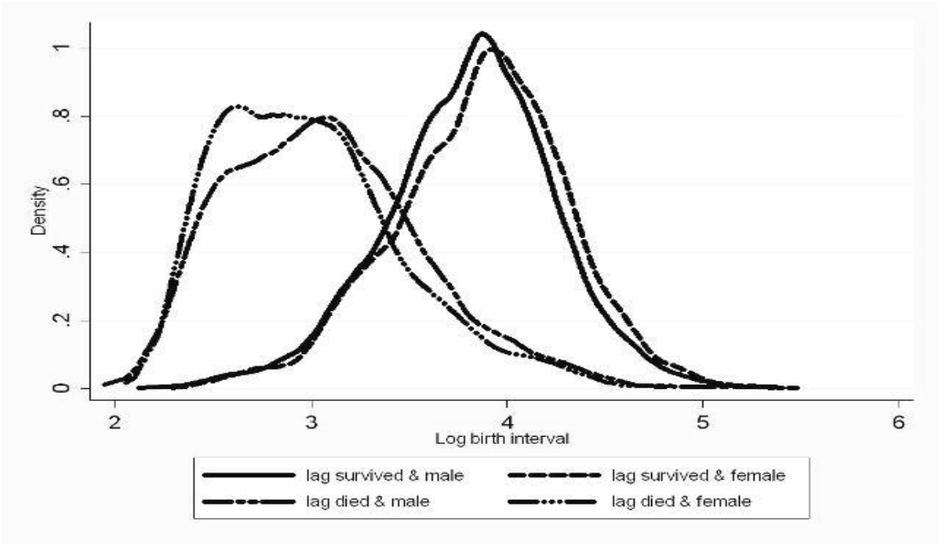


Figure 3: Birth intervals by survival status and gender of previous child, comparison area

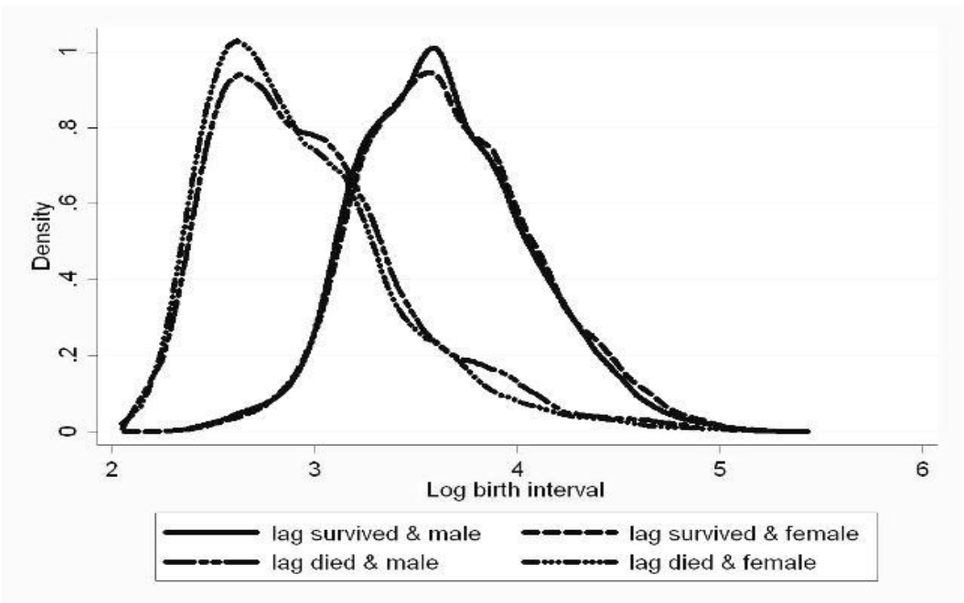


Figure 4: Predicted mortality of index child by survival status of previous child at infancy and log birth interval, ICDDR,B area

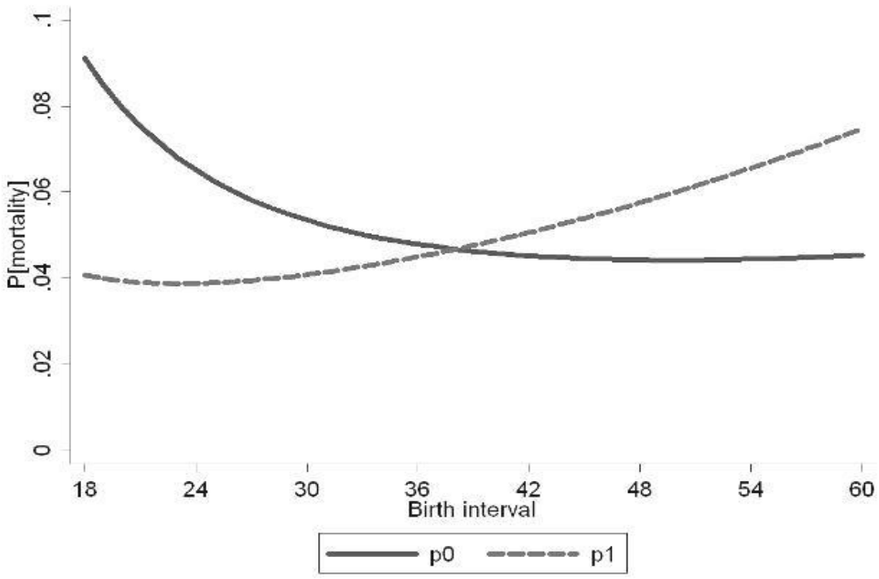
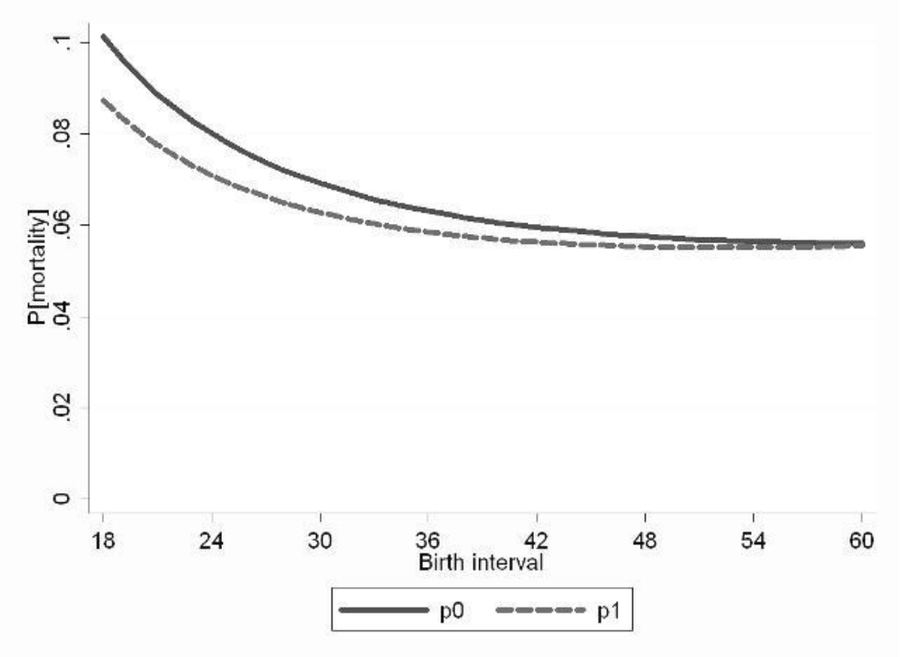


Figure 5: Predicted mortality of index child by survival status of previous child at infancy and log birth interval, comparison area



Chapter 4

Does Family Planning Reduce Infant Mortality? Evidence from Surveillance Data in Matlab, Bangladesh[11]

4.1 Introduction

Family planning programs were initiated for the wellbeing of mother and child. The mechanisms involved relate to family-building patterns like short birth intervals, young (or relatively old) age of the mother at birth, and many births in a short time period. Births that occur at the extremes of maternal age or are preceded by very short birth intervals are at higher mortality risk, as is widely discussed in the demographic literature (see for example, Haaga 1989; Alam and David 1998; Arulampalam and Bhalotra 2006; Omariba et al. 2008; DaVanzo et al. 2008). Through family planning practice a couple can decide the time of birth, the time span between two births, and the (maximum) number of children they want to have. Contraceptive use is a means of family planning and if it leads to a reduction in the proportion of births at these high risks then infant mortality in the population would decline if the level of contraceptive use rises.

Several studies conducted in Bangladesh (and the Matlab region in particular) reveal that the percentage of lower–order births has increased with rising contraceptive use (for example, Koenig et al. 1992; LeGrand and Phillips 1996). By reducing the number of infants born, contraceptive use can enhance the chances for survival: It can, for example, avoid contamination of infectious diseases among closely spaced siblings or reduce siblings' competition for scarce resources such as parental time allocated to child care or the availability of food. Potter (1988) emphasized that proportionally greater reduction of fertility among women whose reproductive health status is poor and the change in family relationships and parenting practices may be crucial ways in which family planning can favourably affect infant mortality.

However, while the theoretical acceptance is wide, empirical evidence on the conjecture that family planning reduces infant mortality is rare. The magnitude of such an effect is also in question. Demographers have different views about the favourable effects of family planning on infant mortality and this thus remains an important issue for further investigation (for reviews see Bongaarts 1987; Trussell 1988; Potter 1988; LeGrand and Phillips 1996). Different explanations

[11] This chapter is joint work with Arthur van Soest, Tilburg University. This paper has been presented in the international conference Population Association of America (PAA) 2011, Washington, D.C, USA. We thank the conference participants and members of committee for their helpful comments and suggestions.

are documented in the literature. Bongaarts argues that the direct effects of contraceptive use on mother's age at birth, birth interval and the number of higher order births are largely offset by the rise in the proportion of high-risk first births, so that the net effect of contraceptive use on infant mortality is small. Secondly, he argues that many of the apparent effects of child-bearing patterns on child mortality are correlated with other factors (see Hobcraft et al. 1985), which needs to be taken into account in the analysis. Several researchers disagree with Bongaarts's first argument. For example, Trussell (1988) argues that Bongaart's analysis is likely to mislead policy makers because the fraction of first births automatically rises due to the total fertility decline, so that the total infant mortality rate at aggregate level is a misleading measure of child health. He emphasizes the need of taking into account the artificial inflation of first-borns when measuring the impact of family planning on infant mortality at the aggregate level. He also argues that the mortality reducing effect of family planning is important among women who use contraception to space their births or to eliminate unwanted high order births.

A recent review conducted by Yeakey et al. (2009) emphasizes the policy relevance of studying the behavioural pathways linking contraceptive use to birth spacing and timing of births and to perinatal and infant mortality. They reviewed fourteen studies, which all find that the use of contraceptives is protective against short birth intervals. This review also points out methodological flaws of the existing studies, which could undermine the accepted rationale for expanding family planning programs to help deliver the maternal and child health benefits of birth spacing. Existing studies typically use retrospective birth-history data collected in cross-sectional surveys, potentially introducing recall bias and heaping of birth intervals at six-month intervals. A few studies investigated either ever-use or never-use of contraceptives by mothers, not considering the timing of contraceptive use in relation to births.

According to this review none of the existing studies used randomized controlled trials to test the effect of contraceptive use on the outcome of interest-infant mortality. Implementing such a design would require not only a very large sample size, but also monitoring continuous episodes of contraceptive use, pregnancies, conceptions, completion of pregnancies, and the morbidity and mortality outcomes of mother and child at least one year postpartum. Indeed, in this regard it seems that an observational design is perhaps inevitable as a feasible alternative. Ideally such a design should be implemented prospectively. Thus, the use of the prospective data from Matlab Bangladesh might be a good alternative for randomized controlled trials (see for example Phillips et al. 1982). Several studies using Matlab data investigated the determinants of infant mortality (DaVanzo and Starbird 1991; Hale et al. 2006; DaVanzo et al. 2007, 2008). However, these studies do not assess explicitly the magnitude of the effect of contraceptive use. Taking into account limitations of all fourteen studies, and in line with the emphasized importance of taking into account correlation and unobserved heterogeneity (Hobcraft et al. 1985; LeGrand and Phillips 1996), the review of Yeakey et al. (2009) concludes that more rigorous modelling is needed, preferably on the basis of longitudinal prospective data. This is exactly where the current study aims to contribute.

In the Matlab ICDDR,B area community health workers through their monthly routine visits record episodes of contraceptive use, pregnancies, conceptions, and the morbidity and mortality outcomes for all children until five years old. Therefore, using longitudinal prospective data from Matlab known to be of exceptional accuracy and completeness, this study first investigates the effects of infant death and other factors (such as socio-economic status or gender composition of the household) on subsequent contraceptive use, and second, the effects of contraceptive use after a birth on birth intervals and infant mortality. Our main analysis is based upon a model with three parts: an equation explaining infant mortality, a model part that explains whether contraceptives are used after a child is born, and an equation explaining birth intervals. (Sterilization is not considered since the mothers in our sample did not initiate sterilization.) Infant mortality is determined by covariates reflecting socio-economic status, age of the mother, gender of the child, etc., but also by the length of the preceding birth interval. The decision to use contraceptives is driven by similar covariates, but also by survival status of the previous child and the family's gender composition. Birth spacing is driven by contraceptive use and other factors.

Each part of the model also incorporates unobserved mother specific heterogeneity, and the various unobserved heterogeneity terms are allowed to be correlated, so that the estimates of the parameters reflecting the causal effects are consistent under general assumptions about the nature of heterogeneity. This makes the model similar in spirit to a recently developed model for birth spacing, fertility, and neonatal mortality in Bhalotra and van Soest (2008). Furthermore, we perform simulations aimed at uncovering the linkage between contraceptive use, birth spacing and infant mortality, taking into account the effect of an increasing fraction of first- born children on the aggregate infant mortality rate.

4.2 Data

4.2.1 ICDDR,B area and interventions

The International Centre for Diarrheal Disease Research, Bangladesh (ICDDR,B) started the Maternal Child Health and Family Planning (MCH-FP) programme in October 1977 in half of the health and demographic surveillance system (HDSS) area in Matlab to assess the extent to which maternal and child health and family planning services can reduce fertility and mortality in a rural population. In this area, formerly known as MCH-FP area and currently as ICDDR,B area, additional health services were provided and data were collected on events like births, deaths, causes of deaths, marriage, divorce, migration-in, migration-out, family planning practice, and a range of health indicators, for a population of 89,350 from 70 villages. The other half of the Matlab area remained under the standard government health systems and is known as comparison area, with a population of 85,596 from 79 villages. The large population of the ICDDR,B area with different levels of intensity and coverage and the relative isolation of villages permitted the designation of four treatment blocks where special services are offered, and of contiguous comparison areas in which demographic dynamics are monitored but the

contraceptive-use history data are absent. The data from the comparison area therefore cannot be used for the purpose of analysis in this paper.

Data have been collected systematically through regular household visits (once every two weeks until January 1998, and once every month since then). These data, in combination with ICDDR,B population censuses and surveys, permit the evaluation of health and family planning services with a degree of accuracy that is rare in low-income settings (LeGrand and Phillips 1996). Analysis of levels and trends and a comparison between the ICDDR,B area and the comparison area shows clear evidence that the MCH-FP intervention reduces fertility and under-five mortality (see for example, LeGrand and Phillips 1996; Hale et al. 2006; DaVanzo et al. 2007, 2008).

4.2.2 Study sample

We analyse a sample of 31,968 singleton live births and 13,232 mothers who continuously lived and gave all their births in the ICDDR,B area. The data cover the period July 1982 to December 2005; the data before 1982 are not (yet) available for this type of research. A similar set of data was used in several companion papers like Saha and van Soest (2011), but this is the first time we also consider the contraceptive use data available after each birth.

4.2.3 Variables and descriptive statistics

The dependent variables in our models are the length of each time interval between births, a dummy for using contraceptives after each birth, and a dummy for infant mortality of each child born alive. The covariates include birth order of the child, gender of the child, and age of the mother at the time of birth of the child; education of the mother is captured by dummy variables; this may proxy the mother's ability to take good care of her children but may also proxy the family's socio-economic status. Education and occupation of the father also reflect the family's socio-economic status. Another family level covariate is religion, which is included because contraceptive practice may vary between the two groups. In Matlab, different patterns of fertility behaviour are observed by religion (Huffman et al. 1987). To control for environmental factors, we include a dummy for access to running drinking water (a dummy for piped drinking water / tube well), and the distance to the nearest health facility (defined as a sub-centre or ICDDR,B hospital).

The average number of children born per mother is 2.42 and 82.7 percent of all mothers in the sample are Muslims. The mean age of mothers at birth is respectively 24.7 years, and the average birth interval is about 48 months with standard deviation 23 months, and about 11 percent birth intervals are shorter than 24 months. 48 percent of all mothers never attended school. On average, mothers residing within 2 kilometres of distance to a health facility and 88 percent of all mothers have access to running water (tube well/pipe water).

During the observation period (July 1982-December, 2005), mothers used contraceptives after about 84 percent of all 31,968 child births. In 11 percent of all cases they did not use

contraceptives, and about in 4.8 percent of all cases the information on contraceptive use was missing (see Figure 1). The missing observations occur for the most recent births because it is too early to observe contraceptive use status. The average duration of contraceptive use is about 31.4 months with a standard deviation 27.9 months. In about 12 percent of all cases, mothers started using contraceptives more than one year after the previous birth (see Figure 1). In about 55 percent of all cases, they started using contraceptives earlier than 12 months after the previous birth and continued until more than 12 months after birth (see Figure 1). These are the cases where mothers were using contraceptives exactly one year after their previous birth.

Among users, 20.67 percent used *pills*, 46.63 percent used *injections*, 4.74 percent used *IUD*, 11.06 percent used *condoms*, 0.43 percent used *sterilization* and 0.81 percent used a *traditional* method.

Table 1 shows that there is a clear positive relationship between contraceptive use and birth interval length. The birth interval until a next birth is about 53 percentage points (from 24 percent to 77 percent) more likely to be longer than 36 months if a mother uses contraceptives at any time after birth (irrespective of starting time and continuation). There is some variation between the birth interval and the contraceptive method used: using injections or condoms is associated with longer birth intervals than using other methods (pill, traditional, IUD; not shown in the Table).

The bivariate relationships between the socioeconomic variables and contraceptive use and infant mortality are given in Table 2. The results are in line with expectations; for example, first births, shorter preceding time intervals between births, mothers younger than 20, and illiterate mothers are particularly disadvantaged in terms of child survival, and also in contraceptive use. For most covariates, the association with child mortality is opposite to that with using contraceptives, but there are some exceptions. For example, although contraception is higher among Hindu than among Muslim families, it is evident that infant mortality is also higher among Hindus. The latter is in line with findings for India; see Bhalotra et al. (2010a,b).

Finally, Table A1 in the annex gives a more detailed picture of the associations between contraceptive use and infant mortality of successive children. First, it shows that contraceptive use after a given birth is much less common when the child that is born dies during its infancy than when it survives its infancy (the contraceptive use rates are 46.4% (=617/1331*100%) and 83.4%, respectively. Second, the infant mortality rate among children born after an interval during which contraceptives have been used is much smaller than the infant mortality rate among births not preceded by contraceptive use (34.0 versus 62.2 per 1000 births). A possible explanation may be that contraceptive use helps to avoid short birth intervals and short intervals lead to larger mortality risk, but alternative explanations are possible, such as common observed or unobserved factors driving mortality and family planning decisions. The econometric model will disentangle these various explanations.

4.3 Model

The model explains infant mortality (that is, whether the child survives its first twelve months or not) of each child born, contraception decisions after each live birth, intervals between live births, and fertility decisions. It builds on the model of Bhalotra and van Soest (2008) but adds the decisions to use contraceptives or not. To be precise, the endogenous variables in the model are the following, with *i* denoting a mother and *t=1,..,*T_i denoting consecutive live births:

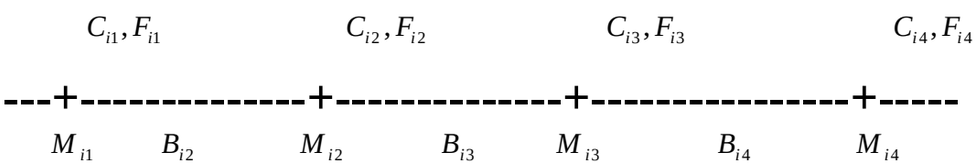
M_{it}: Infant mortality dummy: 1 if child *t* dies; 0 if it survives the first twelve months after birth.

C_{it}: Contraceptive use dummy: 1 if mother *i* uses contraception after giving birth to child *t*; 0 otherwise.

F_{it}: Decision to have another child (1) or not (0).

B_{it}: Log birth interval preceding birth of child *t* (*t>1* only*)*

The sequence of events, which is the basis for the dynamic structure of the model, is illustrated in the following time line:



We do not explain the timing of the first birth (or the decision to use contraceptives before the first birth. The first event we explain is infant survival of the first born child M_{i1}. The second is the decision to use contraceptives or not at any time after birth 1 and (if there is a second birth) before birth 2 (C_{i1}). The information on the exact timing of contraceptive use (starting and ending date) is not very reliable, which is why we only explain the binary decision. Since contraception usually starts at least one year after a live birth, it is a good approximation to treat this variable as an event that takes place when infant mortality of the latest born child is already known. At the same time, the mother also may decide not to have any more children (F_{i1}); this decision is never observed directly, but if a next birth is observed we know that $F_{i1} = 1$.

If $F_{i1} = 1$, a next birth will take place, and if it takes place before the end of the survey window, we observe the birth interval B_{i2}. The second born child can die during infancy or survive, etc.: the sequence of events continues until the mother decides not to have more children ($F_{iT} = 0$) or at the end of the observation period (December 2005).

The model we use is recursive in the sense that each dependent variable may depend on outcomes realized earlier in the sequence of events, but not on future outcomes. Moreover, each outcome may depend on unobserved factors common to all children of a given mother, treated as unobserved individual effects. We use probit equations for the binary outcomes and a regression

equation for the continuous outcomes. Below we discuss the equations for the various outcomes in detail.

Infant mortality

The equation for infant mortality is similar to that in Bhalotra and van Soest (2008). For higher birth orders, a dynamic probit equation with random mother specific effects is used, where the regressors include the preceding birth intervals and variables like the mother's age at birth, which is a function of previous birth intervals: For child t ($t=2,\ldots,T_i$) of mother i, the equation is

$$M_{it}^{*} = X_{it}\beta_m + Z_{it}\gamma_m + \alpha_{mi} + u_{mit} \qquad (1)$$

$$M_{it}=1 \text{ if } M_{it}^{*}>0 \text{ and } M_{it}=0 \text{ if } M_{it}^{*}\leq 0$$

Here X_{it} contains (functions of) the strictly exogenous variables, such as gender of the child, socio-economic status indicators of the household (mother's and father's education, etc.) and characteristics of the village where the household resides. Z_{it} denotes the vector of explanatory variables that are functions of previous outcomes (and are therefore not strictly exogenous). It includes the preceding log birth interval B_{it}, but also (functions of) age of the mother at birth t and, following the literature on scarring (see, for example, Arulampalam and Bhalotra 2006), survival status of the previous child M_{it-1}. The mother specific unobserved heterogeneity term α_{mi} captures unobservable time invariant characteristics influencing the propensity do die of all children in the family. The error term u_{mit} captures idiosyncratic health shocks specific to child t. We assume that the u_{mit} follow a standard normal distribution, independent of each other and of all covariates, and that α_{mi} is normally distributed with mean 0 and variance $\sigma^2_{\alpha m}$ independent of all u_{mit} and X_{it} (but not of Z_{it}).

For mortality of the first child, a separate equation is needed, since in this case there is no preceding birth interval and no preceding mortality outcome. Age at first birth is assumed to be strictly exogenous and can therefore be included in X_{i1}. The equation for infant mortality of child 1 is then given by:

$$M_{i1}^{*} = X_{i1}\beta^{1} + \theta\alpha_{mi} + u_{mi1} \qquad (2)$$

$$M_{i1}=1 \text{ if } M_{i1}^{*}>0 \text{ and } M_{i1}=0 \text{ if } M_{i1}^{*}\leq 0$$

Here β^1 and θ are additional parameters to be estimated and the error term u_{mi1} is assumed to satisfy the same assumptions as the other u_{mit} .

Contraceptive use

We model the observed decisions C_{it} to perform family planning through the use of contraceptives at any time after birth t and (if there is a next birth) before birth $t+1$ (t=1,...,T_i) using the following probit equation:

$$C_{it}^{*} = X_{it}\beta_c + Z_{it}^{c}\gamma_c + \alpha_{ci} + u_{cit} \qquad (3)$$

$$C_{it} = 1 \text{ if } C_{it}^{*} > 0 \text{ and } C_{it} = 0 \text{ if } C_{it}^{*} \leq 0$$

Here X_{it} contains the same strictly exogenous explanatory variables as before. Z_{it}^{c} is the vector of predetermined explanatory variables in this equation, including survival status of preceding sibling and family composition variables (number of surviving girls and boys to mother i). The mother specific unobserved heterogeneity terms α_{ci} capture unobservable time invariant characteristics influencing family planning practice. The error terms u_{cit} capture idiosyncratic errors to the decision of family planning practice after each child birth. We assume that the u_{cit} follow a standard normal distribution, independent of each other and of all covariates, and that α_{ci} is normally distributed with mean 0 and variance $\sigma_{\alpha c}^{2}$ independent of all u_{cit} and X_{it} (but not of Z_{it}^{c}).

Birth-spacing

For a mother who has given births to T_i children, we observe the exact log durations in between two consecutive births $b_{2i}, \ldots, b_{T_i i}$ preceding births $2, \ldots, T_i$. We model these intervals using the following equation:

$$b_{it} = X_{it}\beta_b + Z_{it}^{b}\gamma_b + \alpha_{bi} + u_{bit} \qquad (4)$$

Here X_{it} denotes the vector of strictly explanatory variables, as before. Z_{it}^{b} includes survival status of the preceding sibling, the family composition variables (numbers of surviving girls and boys) and the decision to use contraception C_{it}. The latter captures the mechanism of family through contraceptive use: the use of contraceptives delays the next birth and possibly therefore also reduces the total number of births. The mother specific unobserved heterogeneity term α_{bi} captures unobservable time invariant characteristics influencing the birth interval. The error term u_{bit} captures idiosyncratic errors. We assume that the u_{bit} follow a normal distribution, independent of each other and of all covariates, and that α_{bi} is normally distributed independent of all u_{bit} and X_{it} (but not of Z_{it}).

Fertility decisions and right censoring

There is right-censoring in the data since some mothers will not have completed their fertility at the time of the survey. After the end of the observation window (ultimo 2005), some mothers will still have another birth, and others will not. In principle, this could be captured by the model as it is described until now, with a birth interval after the last observed birth that lasts

longer than till the end of 2005. Following Bhalotra and van Soest (2008), however, the model fit can be improved substantially by adding a separate equation reflecting the possible decisions to stop having children after each birth. The reason why this improves the fit is essentially that it can explain why some mothers who are still of reproductive age have no more births long before the end of the observation window. (We will assume that women are no longer of reproductive age when they reach age 45, an age beyond which very few births are observed in our data). Without the additional equation, this would have to be explained by a very long birth interval.

The equation determining whether the woman continues to have children after birth t ($F_{it}=1$) or not ($F_{it}=0$) is specified as follows:

$$F_{it}^{*} = X_{it}\beta_f + Z_{it}^{f}\gamma_f + \alpha_{fi} + u_{fit} \qquad (5)$$

$$F_{it} = 1 \text{ if } F_{it}^{*} > 0 \text{ and } F_{it} = 0 \text{ if } F_{it}^{*} \leq 0$$

As before, X_{it} denotes the vector of strictly exogenous explanatory variables. The vector Z_{it}^{f} includes survival status of the preceding sibling and family composition variables (based upon the number of surviving girls and boys). The mother specific unobserved heterogeneity term α_{fi} captures unobservable time invariant characteristics influencing the fertility decision after each child birth. The term u_{fit} captures idiosyncratic errors. We assume that the errors u_{fit} are standard normally distributed, independent of each other and of all covariates. The mother specific unobserved heterogeneity terms α_{fi} are normally distributed with mean 0 and variance $\sigma_{\alpha f}^{2}$, independent of all u_{fit} and X_{it}.

The outcome F_{it} is observed only partially. If birth t is not the last birth ($t<T_i$) then we know that the mother has decided not to stop having children, so $F_{it} = 1$. But if $t=T_i$, it is possible that she has decided to stop having children ($F_{it} = 0$), but it may also be the case that the next birth interval extends beyond the end of the observation window ($F_{it} = 1$ and right censoring).

Note that we have neither included contraceptive use as an explanatory variable for the decision to continue fertility, nor the fertility choice as a factor driving contraceptive use. This is because we see these two decisions as taken jointly (and at the same point in time), as illustrated on the time line at the beginning of this section. It is clear that the two decisions are related but modelling the mechanics of the decision process is beyond the scope of this study. Instead, we model two decisions in a reduced form type of way, not including the other decision on the right hand side.[12]

Confounding unobserved factors are controlled for by allowing arbitrary correlations amongst α_{fi}, α_{mi}, α_{bi}, and α_{ci}. We will assume they are drawn from a four-dimensional normal distribution with zero mean and an arbitrary covariance matrix, independent of the X_{it} and all the idiosyncratic error terms.

[12] One might argue that this implies that the error terms in equations (5) and (3) should be correlated. This is an extension we leave for future work.

The five equations of this model (including the initial mortality equation) are estimated jointly using simulated maximum likelihood. Conditional on the random mother specific effects, the likelihood contribution of a given mother can be written as a product of univariate normal probabilities and densities over all births following the order of observed events as indicated on the time line sketched above and accounting for the possibility of right censoring. Since mother specific effects are unobserved, the actual likelihood contribution is the expected value of the conditional likelihood contribution, with the expected value taken over the four random effects. This is a four-dimensional integral, which is approximated using (smooth) simulated ML: Multivariate errors drawn from $N(0,I_4)$ are transformed into draws of the random effects using the parameters of the random effects distribution; the conditional likelihood contribution is then computed for each draw and the mean across R independent draws is taken. If $R\rightarrow\infty$ with the number of mothers N, this gives a consistent estimator; if draws are independent across households and $R\rightarrow\infty$ faster than $\sqrt{N}$, then the estimator is asymptotically equivalent to exact ML (see, for example, Hajivassiliou and Ruud 1994). To reduce the sampling variance in the simulations, we used Halton draws (see Train 2003). The results we present are based on $R=50$. We checked the sensitivity of our parameter estimates for the number of the draws (comparing with larger R) and the nature of the draws (using Halton draws with different seeds) and always got very similar results, as in chapter 3. The estimation procedure is very similar in spirit to the one used by Bhalotra and van Soest (2008); see also their online appendix for details.

4.4 Estimation results

Table 3 reports the parameter estimates, using the benchmark definition of contraceptive use. The estimates of the specification with an alternative definition of the contraceptive use dummy are presented in Table A2 in the appendix. In the discussion in this section we focus on the benchmark specification; the results in Table A2 will be discussed in Section 6. The top panel of the Table presents the estimates of the parameters in the four main equations; the bottom panel shows the estimates of the covariance structure of the unobserved heterogeneity terms. Estimates for the static equation for mortality of the first child are available upon request; they are very similar to those in Saha and van Soest (2011).

Contraceptive use

The estimates in the contraceptive use equation are in line with existing results on the determinants of contraceptive use in rural Bangladesh; see for example, Koenig et al. (1992) or Rahman et al. (1992). Acceptance of contraception is significantly (at the 5 percent level) higher among Hindus than among Muslims, in line with the bivariate relation in Table 2. The estimated difference in the probability to use contraceptives keeping other observed and unobserved characteristics constant is about 1.7 percentage points.[13] Contraceptive use is increasing in both maternal and paternal education, with larger effects of paternal education. The strong association

[13] Estimated marginal effects (keeping other observed and unobserved characteristics constant) for the average observation are about 0.19 times the corresponding parameter estimate.

with parental education levels is in line with Rahman et al. (1992, Table 1), while Koenig et al. (1992, Table 3) find a much weaker relation with maternal education in the ICDDR,B area. If the father is a day-labourer, however, contraceptive use is significantly more likely, which is not in line with the bivariate relationship in Table 2. Perhaps these families have a larger tendency to postpone having more children until the socio-economic position of the breadwinner improves. The likelihood of contraceptive use is increasing in the mother's age at birth, a common pattern in developing country data. Mothers of later birth cohorts exhibit a significantly increasing trend of contraceptive use.

The death of the last born child at infancy substantially reduces the likelihood of contraceptive use (by about 17 percentage points in the benchmark specification), in line with the *replacement hypothesis* that families want to replace a lost child. This is widely regarded in the demographic literature (for example, see Rahman et al. 1992 or Bhalotra and van Soest 2008). The effects of the numbers of surviving boys and girls are consistent with son preference: having at least one boy has a somewhat stronger (positive) effect on the decision to use contraceptives than having at least one girl (the marginal effects are about 10.0 and 8.4 percentage points), and each additional son in the family increases the likelihood of contraceptive use more than each additional daughter (with, for the average observation, about 6.7 and 3.4 percentage points, respectively). Similar conclusions concerning son preference in family planning have been drawn in other studies for Bangladesh (see for example, Rahman et al. 1992; Koenig et al. 1992).

Birth intervals

The parameter estimates in the log birth-spacing equation show that, keeping constant other factors including the decision to use contraceptives at any time after the previous birth, birth intervals tend to be shorter for high birth orders, which is consistent with the stylized fact that short birth intervals are associated with high fertility. Mothers with more education consistently have longer birth intervals. Birth spacing is increasing in maternal age. In more developed villages with piped/tube well water, birth intervals are longer.

As expected, using contraceptives leads to a large and significant increase in the space between births – it increases the interval by around 60 percent (exp(0.495)-1)*100%). On the other hand, keeping contraceptive use and other factors constant, death at infancy of the previous child shortens the subsequent interval between births by 43 percent (exp(-0.55)-1)*100%), in line with the replacement hypothesis. The effects of the surviving numbers of boys and girls are again consistent with son preference: having a boy increases the birth interval by twice as much as having a girl, and each additional boy has a much larger effect than each additional girl. These findings are consistent with the earlier findings in the contraceptive equation. These results show that the decision to use contraceptives and the length of the birth interval conditional on the decision to use contraceptives (which will depend upon starting and ending date of contraceptive use, which are not explicitly modelled) are both determined by similar family planning considerations.

Infant mortality

The parameter estimates in the mortality equation in Table 3 are largely in line with the general conclusions about the determinants of infant mortality in developing countries (see Bhalotra and van Soest 2008; Omariba et al. 2008) and our findings in chapter 2 or in Saha and van Soest (2011). A difference compared to our earlier study (chapter 2) is that we find that the effect of lagged mortality on the probability of infant death of the index child is negative but insignificant, where in chapter 2(Table 5) this effect was negative and significant at the 5 percent level when the birth interval was controlled for as an exogenous covariate. This small and insignificant parameter estimate suggests that a negative learning effect is compensated by a positive scarring effect through, for example, depression induced by the previous infant's death. We allow for a nonlinear relation between birth intervals and infant mortality. The estimates imply that mortality risk falls with the length of the birth interval over most of the relevant range of birth intervals (until about 57 months), a finding which is in line with the existing literature (see, for example, Rutstein, 2005, or Conde-Agudelo et al., 2006). Taking account of the nonlinear relation between birth spacing and infant death, we find that at the average birth interval length, an increase of the birth interval by 10 percent reduces mortality by about 0.11%-points. Since the effect of contraceptive use on the log birth interval is about 0.495, this implies that, for the average observation, using contraceptives reduces the mortality probability by about 5.4 deaths per 1000 live births.

Fertility

The final column of Table 3 presents the estimates of the auxiliary fertility equation explaining whether, after each birth and mortality outcome, a family decides to have another birth or not. Fertility falls with the level of education of both mother and father, with mother's education having the larger effect. Muslims show a higher tendency to continue fertility than their Hindu counterparts, and this finding is consistent with contraception differentials by religion. It is less clear why, keeping other factors constant, the probability to have another birth is highest among the youngest birth cohort of mothers and increases with the mother's age at previous birth. In villages with access to running water (tube well or piped water) mothers are less likely to continue their fertility. The family composition variables again show evidence of son preference in family planning, consistent with the findings in both the contraceptive use and the birth spacing equation.

Unobserved heterogeneity

The bottom panel of Table 3 describes the estimated covariance structure of the unobserved heterogeneity terms. (The covariance matrix is specified as $\Lambda\Lambda'$ for a positive semi-definite lower triangular matrix Λ; the estimated auxiliary parameters are not presented to save space) Unobserved mother specific heterogeneity is large and significant in the contraceptive and fertility equations, reflecting 33 percent and 44 percent (denoted in the table by ρ), of the total unsystematic variation (for given values of the observed covariates and endogenous explanatory variables in each equation), compared to only 7 percent in the mortality and birth interval equations.

We find a large negative correlation between the unobserved heterogeneity terms in the fertility and contraceptive use equations, and between the fertility and birth spacing equations. This suggests that, keeping observed factors constant, mothers who desire more children are less likely to use contraceptives ($\text{corr}(\alpha_{fi}, \alpha_{ci})$ = -0.73), and anticipate this by reducing birth-spacing ($\text{corr}(\alpha_{bi}, \alpha_{fi})$ = -0.76). This is consistent with target fertility models discussed in, for example, Wolpin (1997). The negative correlation of the unobserved heterogeneity terms in the mortality and contraceptive equations (-0.31) suggests that mothers whose children have relatively high mortality risks respond to this by planning more children and not using contraceptives. This interpretation contradicts, however, the (modest) positive correlation between unobserved heterogeneity terms in the mortality and birth spacing equations (+0.21).

4.5 Simulations

To illustrate the importance of family planning for birth spacing, fertility, and infant mortality, we performed some simulations, in a similar way as the simulations in Bhalotra and van Soest (2008, Table 3) and as in chapter 3. These simulations show the benefits of family planning programs that delay second births through lengthening birth intervals and avoid high risk births in the young birth cohorts of mother. It illustrates the main novelty of our approach – the fact that our model incorporates various mechanisms that lead to associations between family planning, birth spacing, fertility, and mortality outcomes, accounting for the effects of endogeneity in contraceptive use decisions, timing of births (and therefore also age at birth etc.), birth intervals, and mortality risks.

The simulations use the covariates (including, for example, date of first birth) as observed for each mother in the actual sample. We then generated, for each mother in the sample, unobserved heterogeneity terms, error terms, and new outcomes (the dependent variables in our model) using the estimated parameters of each equation. The outcomes were generated recursively, using the timing of the events as sketched in Section 4.3. For example, for a given mother, we take the date of first birth as given and first generate the mortality outcome of the first child (using equation (2)). Given simulated mortality, we then generated the contraceptive use decision and the fertility decision after the first birth (equations (3) and (5)). If the fertility decision is positive, we then generate a birth interval, and update calendar time and age of the mother at the second birth. Given these variables and the other covariates and the previous mortality outcome, we then generate the mortality outcome of the second born child, etc. In this way we generate complete contraceptive use, fertility, and mortality patterns for each mother in the sample. To reduce simulation variance, this is repeated 25 times for each mother.[14]

Column 1 summarizes the simulation outcomes according to a benchmark simulation where all mechanisms at work in the estimated model are active. This simulation reproduces the

[14] The simulations take the parameter estimates as given. In principle, it would be possible to compute a standard error for each simulation outcome by repeating the simulations for other draws of the model parameters from their estimated (asymptotic) distribution but this would require a substantial computational effort.

means in the raw data, showing that the model is able to reproduce these basic features of the data. This simulation also reproduces the substantial difference between mortality of first born children (simulated at 67.2 per 1000 live births) and mortality of higher order births (39.6 per 1000 births on average; 40.4, 37.9, 39.0, 42.2 and 46.8 per 1,000 live births for birth orders 2, 3, 4, 5 and 6, respectively).

The other columns present the deviations from the benchmark considering the two hypothetical extreme cases of contraceptive use: in column 2, everyone is always assumed to use contraceptives. In column 3, no contraceptives are used at all. The latter is a more dramatic change compared to the benchmark than the former, since in the benchmark simulation contraceptives are used in 88.6 percent of all cases.

The simulation in column 2 shows that, according to our model estimates, if contraceptives were used after each birth, the average birth interval length would increase by about four months. Since short birth intervals are then more often avoided, it would also reduce the mortality risk of higher order births. The estimated reductions in mortality of children of birth order two and higher would be substantial: 7.9 percent for all birth orders of 2 and higher. It is particularly large for birth order 2 with a reduction of 9.2 percent (3.7 per 1000 live births – from 40.4 to 36.7; these figures are not shown in the table). The longer birth intervals would also reduce total fertility, by about 2.4 percent. This implies that the weight of first born children in the total infant mortality rate will increase. Since the infant mortality risk for first born children is higher than for higher birth orders (and since contraceptives do not affect this mortality rate), this composition effect tempers the favourable effect of contraceptives on survival chances of higher order births: the total infant mortality rate still falls compared to the benchmark situation, but by much less than the mortality rate for higher birth orders.

Results by maternal education level (not shown in the table) show that the benefits of complete contraceptive use would be particularly large for the lowest socio-economic status group, mothers without any education. This is because they have the lowest contraceptive use in the benchmark situation (84.4% compared to 88.6% for the complete sample) but also because they have shorter birth intervals and the most vulnerable children (their infant mortality rate among children of birth order 2 and higher is 49.5 per 1000 births in the benchmark situation, compared to 39.6 per 1000 for the complete sample). If everyone would use contraceptives after each birth, birth interval lengths in the no education group would increase by 6 months on average, and infant mortality among higher order births would fall by 8.7% (compared to 7.9% for the complete sample). Their total infant mortality rate would fall by 3.2% (2.9% for the whole sample).

The simulation in column 3 indicates that, if contraceptives were never used after any birth, the average birth interval length would shorten by more than 13 months, and this would raise the mortality risks in higher order births by 10.6 percent (from 39.6 to 43.8 per 1000 live births). Particularly for second order births the effect would be large (6.3 per 1000). The shorter birth intervals lead to an increase of the total fertility rate by 19.3 percent, implying that the

weight of first born children in the total infant mortality rate will fall. This leads to a negative composition effect on the total infant mortality rate, that almost completely compensates for the rise in mortality of higher order births – the total infant mortality rate increases by 1.6 percent only (from 51.2 to 52.1 per 1000 live births) compared to the benchmark situation.

4.6 Alternative model specification

Table A2 presents the estimation results for the alternative definition of the contraceptives use dummy – considering whether a mother uses contraceptives at a specific point in time: exactly 12 months after a given birth. Most of the parameter estimates are similar to those in Table 3, but there are exceptions, particularly, as expected, in the coefficients of the contraceptives equation. Muslim mothers are much less likely to use contraceptives after exactly one than Hindu mothers and the difference is now significant at the 5 percent level. Contraceptive use after one year also increases significantly with birth order. On the other hand, mother's education plays a much smaller role than in Table 3. The effect of lagged mortality is still somewhat stronger than in Table 3, but, unexpectedly, the effects of the family composition variables (surviving boys and girls) are much smaller and less significant. These variables still have the expected strong and significant effects on birth intervals and fertility decisions, but not on the decision to use contraceptives at the chosen specific point in time.

The effect of contraceptive use defined in this alternative way on birth spacing remains positive and significant, but is much smaller than in Table 3 (0.341 instead of 0.495), suggesting that contraceptive use after exactly 12 months does not capture the full effect of contraception decisions on birth spacing; this is the main reason why we prefer the definition of using contraceptives at any time instead of the alternative.

Table 5 gives the results of the simulations discussed in the previous section for the estimated model in Table A2, using the alternative dummy on contraceptive use. The benchmark simulation predicts that contraceptives are used at exactly one year after birth in 58 percent of all cases. The other outcomes of the benchmark simulation are similar to those in Table 4 and reproduce the corresponding statistics in the sample.

The simulation in column 2 of Table 5 shows that, according to our alternative model estimates, if everyone always used contraceptives at one year after each birth, the average birth interval length would increase by about 7.5 months. The effect seems larger than in Table 5, but that is because the change from not using to using contraceptives affects many more cases now (42 percent rather than 12 percent). As a consequence, mortality of children of birth orders two and higher would fall by 6.9 percent (from 39.4 to 36.7 per 1000 live births). Again, the reduction is relatively high for second order births (8.2 percent; not shown in the table). The longer birth intervals would reduce total fertility by about 6.0 percent. This implies that the weight of first born children in the total infant mortality rate will increase. This composition effect tempers the favourable effect of contraceptives on survival chances of higher order births

if the total infant mortality rate is considered: the total infant mortality rate falls by only 1.5 percent (from 51.0 to 50.3 per 1000 live births) compared to the benchmark situation.

Column 3 of Table 5 gives the simulation results when no one would use contraceptives one year after birth. The average birth interval length would fall by about 5.7 months. As a consequence, mortality of children of birth orders two and higher would rise by a modest 1.4 percent (3.8 percent for children of birth order 2, but smaller effects for higher birth orders), while total fertility would increase by 8.6 percent. This induces a negative composition effect on total infant mortality including first children since the weight of relatively vulnerable first born children falls. This composition effect is larger than the direct effect through birth intervals so that the sum of the two effects is also negative: total infant mortality falls by 1.1 percent compared to the benchmark situation.

4.7 Discussion and conclusion

Several studies using Matlab data investigated the determinants of infant mortality (DaVanzo and Starbird 1991; Hale et al. 2006; DaVanzo et al. 2007 and 2008). However, these studies did not assess explicitly the magnitude of the effect of family planning programs on birth intervals and thereafter on infant mortality. The major motivation of our current study is the conclusion drawn by Bongaarts (1987) and a recent review paper by Yeakey et al. (2009). We use the prospective pregnancy-history data from Matlab, Bangladesh where community health workers (CHWs) through their monthly routine visit record episodes of contraceptive use, pregnancies, conceptions, and morbidity and mortality outcomes for mothers and children younger than five.

Exploiting dynamic econometric panel data modelling, our analysis allows for taking into account endogeneity of birth intervals in the mortality equation, reverse causality of mortality and fertility (probability of having further birth), and identifies the causal effect of contraceptive use on birth intervals.

The covariate effects on infant mortality, contraceptive use, and birth intervals are generally in line with expectations and associations observed in the existing demographic literature. Some remarkable findings are: contraceptive use after a given birth is likely to increase the length of log birth intervals by about 60 percent Feeding this effect in the mortality equation shows that it is also likely to reduce the effects of maternal depletion in child births. We find evidence of son preference in both contraceptive use decisions and log birth interval choices conditional on using contraceptives or not.

Contraceptive use may be related to breastfeeding, since breastfeeding also can delay a new pregnancy. The effects of breastfeeding on birth intervals and childhood deaths in the literature are mixed; see, e.g., Smith (1985) and Retherford et al. (1989). We have investigated the associations of breastfeeding with birth intervals and, surprisingly, found no significant differences in birth spacing by breastfeeding status. However, a large and significant difference in birth spacing by contraceptive use status exists and this difference does not vary by breast feeding status. This finding is in line with van Ginneken (1974) who found that lactation is less

adequate as a birth spacing method than modern contraceptives. Still, it can be seen as a limitation of our study that we did not explicitly incorporate breastfeeding in our model.

Our findings are in line with the argument of Bongaarts (1987) that the direct effects of contraceptive use on mortality are largely offset due to changes in the composition of births by age, birth order, and birth interval, particularly the rise in the proportions of high-risk first births (see also Hale et al. 2009). These effects are disentangled in the simulation analysis. It shows that, as Bongaarts argued, the net effect of family planning on reducing total infant mortality is small. At the same time, the results confirm the favourable effects of family planning programs on child survival for second and higher birth orders that work through birth spacing – and our simulations imply that further increase of contraceptive use has the potential or reducing infant mortality among second and higher order births by about 7.9 percent. (11 infant deaths per 1000 live births). This leads to the policy implication that strengthening family planning programs helps to reduce infant mortality. Since this is particularly the case for lower socio-economic groups, it also improves equity across socio-economic groups.

In our analysis, the date of the first birth is given and not explained. Children of very young mothers (age at birth less than 20 years) have a much larger risk of infant mortality and thus it remains important for further study to analyze how contraceptive use plays a role in increasing age at first birth. This information will be important for strengthening family planning programs for newly married couples. Increasing the age at first birth may also lead to fertility reductions through reducing the total reproductive span of women, something that is already on the policy agenda.

Our current analysis has several other limitations. Due to availability of data we could not model the decision to discontinue the use of contraceptives and the births that are due to such discontinuation or failure of the contraceptive method. That these events are common in Bangladesh is known from contraceptive use history data (see Steele and Diamond 1999; Bairagi et al., 2000; Saha et al., 2004). Saha et al. (2004), for example, estimate that about 50% of all mothers discontinue using a contraceptive method within two years of initiating it, and discontinuation is particularly large for pills and condoms. Different rates are found in other studies that use the calendar data from the Bangladesh Demographic and Health Surveys (BDHS), where injection users are more likely to discontinue than pill or condom users. This is possible because the method mix observed in the nationally representative BDHS is different from that of Matlab. A study conducted in Matlab by Bairagi et al. (2000) found that the cumulative probability of first method failure within one year of method acceptance during 1990-1994 was, for example, 12.9% for pills and 22.0% for condoms. Our alternative specification is a first crude attempt to take account of how long contraceptives are used instead of just whether they are used or not. Future research can look at the timing in more detail.

Moreover, our model uses one dummy of contraceptive use and does not distinguish between the various methods, avoiding the need to complicate the model further with the choice

of method in an already intricate model. Modelling the choice of the type of contraceptives may give more insight in the effectiveness of method-mix in lengthening birth intervals, and thus seems interesting topic for future research.

Finally, it would be interesting to extend the current analysis to a setting without extensive health and family planning services such as the comparison area in Matlab. This can disentangle the effects of family planning programs on the duration of birth intervals and on infant mortality in a society where only government health services are available and contraceptive use is less prevalent.

Tables

Table 1. Distribution of contraceptive use and birth intervals.

Birth interval	<-24 months	25-36 months	37 + months	Total (N)
Contraceptive Use		Row Percentage		
No	41.13	35.03	23.84	3,460
Yes	6.47	16.58	76.94	14,472
Total (N)	2,360	3,612	11,960	17,932

Notes: observations on contraceptive use (after each birth) are missing for total 1,524 birth records where 804 birth records after first-borns and excluded from this analysis, and due to first-borns 13,232 observations are excluded from this analysis.

Table 2. Descriptive statistics of different predictors of infant mortality and contraceptive use in Matlab, Bangladesh, birth cohort 1982-2005.

	Children (%)	Infant mortality (%)	Contraceptive use after birth (%)
Birth order			
1	41.39	6.70	79.46
2-3	46.55	3.86	86.39
4+	12.06	4.28	89.68
Gender of child			
Male	16,294	5.43	84.43
Female	15,674	4.73	83.38
Preceding birth interval (excluding first-born)			
<=24 months	7.61	7.27	
25-36 months	11.71	4.12	
37+ months	39.29	3.26	
Mother's age at birth (years)			
<=19	10.89	8.13	79.89
20-24	47.03	5.55	81.60
25-29	22.23	3.79	86.26
30-34	16.23	3.70	88.98
35+	3.62	4.06	87.04
Religion			
Muslim	83.03	4.97	83.72
Not Muslim	16.97	5.68	84.86
Maternal education level			
No education	48.48	6.28	81.84
At least primary education	24.86	4.53	86.32
At least secondary education	26.66	3.44	85.45
Mother's birth cohort			
Before 1966	6,304	6.44	74.11
1966-1970	9,416	5.62	83.60
1971-1975	7,306	4.71	89.46
1976+	8,942	3.88	86.62
Paternal educational level			
No education	55.67	5.53	80.59
At least primary education	22.65	5.58	86.98
At least secondary education	21.68	3.43	89.25
Paternal occupation			
Day laborer	19.61	7.53	77.21
Not day laborer	80.39	4.49	85.55
Source of drinking water			
Pipe/tube-well	87.76	4.68	85.15
Other	12.24	8.0	75.03
Distance to nearest health facility			
≤ 1 km	35.80	4.97	84.84
1-2 km	42.44	5.06	83.72
>2 km	21.76	5.32	82.78

Table 3: Estimation Results.

Variable	Contraceptive use		Log birth space		Infant mortality		Prob (next birth)	
Panel A	parameter	s.e	parameter	s.e	parameter	s.e	Parameter	s.e
Male	-0.070	0.041	0.002	0.010	0.025	0.037	-0.031	0.045
Muslim	-0.092*	0.042	-0.008	0.010	-0.023	0.052	0.602**	0.064
Birth order	0.057	0.071	0.084**	0.018	0.064	0.103	-0.072	0.086
Birth order square	-0.011	0.010	-0.019**	0.002	-0.007	0.014	0.005	0.009
Mother's age at birth (years)/10	0.005*	0.002	0.003**	0.001	-0.012**	0.003	-0.001	0.003
Mother's age at birth/10 square	0.230**	0.047	0.048**	0.012	0.193**	0.060	0.228**	0.059
Maternal education level								
At least primary education	0.116**	0.037	0.027**	0.008	-0.047	0.047	0.024	0.050
At least secondary education	0.181**	0.045	0.043**	0.010	-0.023	0.060	-0.366**	0.071
Mother's birth cohort								
1966-1970	0.565**	0.038	-0.007	0.009	-0.026	0.047	0.033	0.046
1971-1975	1.208**	0.0467	0.017	0.011	-0.133*	0.058	0.007	0.064
1976+	1.841**	0.057	0.052**	0.012	-0.159*	0.069	0.574*	0.258
Paternal educational level								
At least primary education	0.200**	0.037	-0.024*	0.008	0.056	0.044	-0.011	0.050
At least secondary education	0.291**	0.041	-0.024*	0.009	-0.205**	0.059	-0.129*	0.056
Father is day labourer	0.194**	0.052	-0.022	0.011	0.131*	0.055	-0.508**	0.064
No tubewell/piped water	0.052	0.037	0.031**	0.009	-0.163*	0.056	-0.100	0.053
Distance to health centre (in km)	-0.009	0.015	0.006	0.003	0.005	0.020	-0.018	0.017
Lagged contraceptive use			0.495**	0.010				
lagged infant mortality	-0.880**	0.060	-0.554**	0.016	-0.020	0.072	-0.139	0.089
First boy surviving	0.528**	0.062	0.126**	0.016			-0.955**	0.105
First girl surviving	0.440**	0.063	0.085**	0.016			-0.873**	0.096
After first boy, # of boys surviving	0.353**	0.056	0.079**	0.015			-0.678**	0.085
After first girl, # of girls surviving	0.178**	0.052	0.027	0.015			-0.298**	0.072
Preceding log birth interval					-1.586**	0.373		
Preceding birth interval square					0.196**	0.052		
Constant	-0.226	0.293	2.899**	0.073	3.131**	0.750	4.472**	0.462
Panel B								
P	0.328		0.067		0.066		0.435	
Correlation (row1)			0.184		-0.306		-0.727	
Correlation (row2)							-0.764	
Correlation (row3)			0.205				-0.153	

Notes: * 2 < t-value < 3; ** t-value ≥ 3

Reference category: gender is female, religion is Muslim, mother and father have no education, father is not day-labourer, source of drinking water is tube-well/pipewater, and mother's birth cohort before 1966.

Table 4. Simulation results.

	Column 1	Column 2	Column
	Benchmark	**Contraceptive**	**Contraceptive non-**
Birth interval	43.82 months	+4.13 months	-13.25 months
Number of births (fertility)	2.37	-2.43%	+19.36%
Number of survivors	2.25	-2.28%	+19.26%
For all children			
Infant mortality	51.3/1000	-2.88%	+1.62%
For children after first born			
Infant mortality	39.6/1000	-7.86%	+10.61%

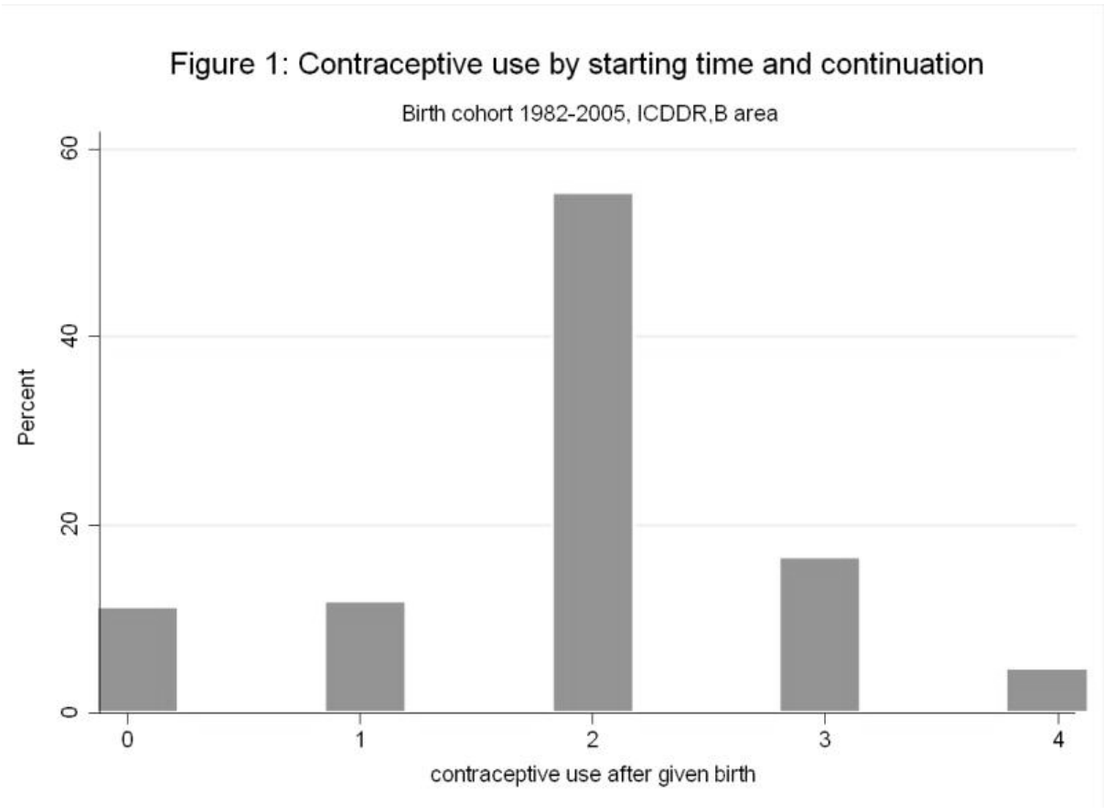
Note: column1: Outcomes benchmark simulation, columns 2 and 3: deviations from the benchmark simulation if everyone uses contraceptives (column 2) or noone uses contraceptives (column 3)

Table 5. Simulation results (alternative definition of contraceptives use).

	Column 1	Column 2	Column 3
	Benchmark	**Contraceptive use**	**Contraceptive non-**
Birth interval	43.43 months	+7.53 months	-5.67 months
Number of births (fertility)	2.41	-6.01 %	+8.65 %
Number of survivors	2.28	-5.93 %	+8.72 %
For all children			
Infant mortality (%)	51.0/1000	-1.48 %	-1.13 %
For children after first born			
Infant mortality (%)	39.4/1000	-6.89 %	+1.44 %

Note: column1: Outcomes benchmark simulation, columns 2 and 3: deviations from the benchmark simulation if everyone uses contraceptives (column 2) or noone uses contraceptives (column 3)

Figure



Note: 0 = non-use
1 = start one year after birth or later
2 = start within one year after birth and continue until more than one year after birth
3 = start within one year after birth and stop before one year after birth
4 = missing

Annex

Table A1. Infant mortality of previous child, Contraceptive use, and Infant mortality of the next child.

	Previous child died	Previous child did not	All children
Contraceptive	Infant mortality next child	Infant mortality next	Infant mortality
Yes	81.0/1000 (n= 617)	32.0/1000 (n=14,482)	34.0/1000
No	77.7/1000 (n= 734)	58.3/1000 (n= 2,884)	62.2/1000
	79.2/1000 (n=1,351)*	36.4/1000	39.5/1000

Notes: observations on contraceptive use (after each birth) are missing for total 1,524 birth records where
*2 birth records after first-borns and excluded from this analysis. ** 17 birth records after first borns and excluded from the analysis
- Due to first-borns 13,232 observations are excluded from the analysis, and the remaining missing birth records are in the first obsrevations

Table A2. Estimation results with alternative definition of contraceptive use.

Variable	Contraceptive use		Log birth space		Infant mortality		Prob (next birth)	
Panel A	parameter	s.e	parameter	s.e	Parameter	s.e	parameter	s.e
Male	-0.091**	0.027	0.003	0.010	0.024	0.036	-0.041	0.041
Muslim	-0.188**	0.026	-0.008	0.010	-0.021	0.050	0.491**	0.050
Birth order	0.211**	0.052	0.081**	0.020	0.060	0.101	-0.044	0.084
Birth order square	-0.006	0.005	-0.018**	0.002	-0.007	0.014	0.006	0.008
Mother's age at birth (years)/10	0.002	0.001	0.002**	0.001	-0.011**	0.003	-0.002	0.003
Mother's age at birth/10 square	0.060*	0.026	0.043**	0.012	0.178**	0.060	0.186**	0.052
Maternal education level								
At least primary education	0.043	0.025	0.030**	0.008	-0.047	0.045	0.029	0.042
At least secondary education	0.062*	0.028	0.053**	0.010	-0.023	0.058	-0.312**	0.057
Mother's birth cohort								
1966-1970	0.337**	0.028	0.015	0.009	-0.023	0.045	0.042	0.039
1971-1975	0.656**	0.032	0.050**	0.011	-0.133*	0.057	0.035	0.056
1976+	0.688**	0.034	0.079**	0.013	-0.148*	0.067	0.208	0.141
Paternal educational level								
At least primary education	0.168**	0.025	-0.022*	0.008	0.056	0.042	0.025	0.042
At least secondary education	0.237**	0.026	-0.024*	0.009	-0.200**	0.057	-0.070	0.047
Father is day labourer	0.238**	0.032	-0.034*	0.012	0.128*	0.053	-0.397**	0.050
No tubewell/piped water	0.175**	0.029	0.031**	0.009	-0.165*	0.055	-0.126*	0.046
Distance to health centre (in km)	-0.007	0.010	0.005	0.004	0.005	0.019	-0.016	0.014
Lagged contraceptive use			0.341**	0.008				
lagged infant mortality	-1.109**	0.057	-0.574**	0.017	0.007	0.069	-0.141	0.081
First boy surviving	0.117*	0.050	0.146**	0.018			-0.765**	0.094
First girl surviving	-0.014	0.049	0.103**	0.018			-0.702**	0.087
After first boy, # of boys surviving	0.0001	0.044	0.092**	0.016			-0.536**	0.078
After first girl, # of girls surviving	-0.082*	0.042	0.032*	0.016			-0.218**	0.069
Preceding log birth interval					-1.627**	0.365		
Preceding birth interval square					0.203**	0.051		
Constant	-0.587**	0.175	3.082**	0.075	3.103**	0.735	3.961**	0.381
Panel B								
ρ	0.225		0.015		0.028		0.242	
Correlation (row1)			0.025		-0.439		-0.489	
Correlation (row2)							-0.811	
Correlation (row3)			0.086				-0.168	

Notes: * 2 < t-value < 3; ** t-value ≥ 3
Reference category: gender is female, religion is Muslim, mother and father have no education, father is not day-labourer, and source of drinking water is tube-well/pipewater, mother's birth cohort before 1966.
Alternative specifications: refer contraceptive use=1 if mother initiated method use within one year after birth and continued until at least one year after birth, otherwise contraceptive use=0.

Chapter 5

Cause-specific neonatal deaths: levels, trend and determinants in Rural Bangladesh, 1987-2005[15]

5.1 Introduction

Of the 130 million children born alive each year worldwide, about four million die in the first four weeks after birth. 99% of these deaths occur in low and middle income countries; 4% occur in Bangladesh (Lawn et al. 2005). Achieving the fourth Millennium Development Goal (MDG-4) of reducing under-5 child mortality by two-thirds between 1990 and 2015 remains one of the United Nations' global priorities (United Nations 2001). The recent trends in mortality suggest that without substantial reductions in neonatal mortality, MDG-4 will not be achieved (Lawn et al. 2005). Global reviews suggest that almost 60% of childhood deaths can be prevented by increasing the coverage of existing newborn and child health interventions (Jones et al. 2003). The information on causes of neonatal and child death is important here, since - it can be used to prioritize and to increase the effectiveness of disease-specific interventions (Baqui et al. 2001; Lawn et al. 2006).

Reducing neonatal mortality is a particularly important issue in Bangladesh. Although child mortality rates in Bangladesh declined sharply during the last decades of the previous century, the reduction is slowed down particularly in the neonatal period. Among child deaths, those that occur during the first month represent an increasing proportion. Estimates based upon the Bangladesh Demographic and Health Survey (BDHS) suggest that 70% of all under-five deaths occur in the first year of life and 80% of these occur in the neonatal period (see Figure 1). It is therefore a significant challenge to reduce neonatal mortality in order to meet MDG-4 of reducing under-five mortality from 133 per 1,000 live births to two-thirds between 1990 and 2015. This study analyzes the levels and the trend of cause-specific neonatal deaths in Bangladesh and associated risk factors, both observed and unobserved. The findings may help to design policies that reduce neonatal mortality in Bangladesh in particular, but also are potentially relevant for many other countries in the developing world.

[15] This chapter is joint work with Arthur van Soest, Tilburg University and Govert E. Bijwaard, Netherlands Interdisciplinary Demographic Institute (NIDI). We thank the members of committee for their valuable comments that helped to improve the paper.

We estimate a flexible competing risks model of causes of death until 28 days after birth (neonatal deaths), considering children who survive the neonatal period as censored observations. Our modelling approach is more flexible than in many existing studies of determinants of causes of deaths. It combines a piecewise constant baseline hazard with proportionality assumptions concerning the influence of observed and unobserved risk factors for each cause of death. The model allows the unobserved heterogeneity components in the hazard rates for the various causes of death to be correlated.

Our estimations are based upon prospective panel data from the Matlab region in Bangladesh, following mothers and children over time from 1987 until 2005. Two sets of villages are covered: an intervention area with non-standard health services (International Centre for Diarrhoeal Disease Research, Bangladesh or ICDDR,B area), and an area with standard government-provided health care facilities (comparison area); the differences between the two areas give insight into how the additional health care services shape the child health epidemiology over the period.

5.2 Background

The disease structure of neonatal deaths is different from that of post-neonatal deaths or deaths of children older than one year (Bhatia 1989). Neonatal deaths are associated with biological characteristics of the mother and with problems during pregnancy and child birth, which can be improved by targeted interventions such as tetanus toxoid to pregnant women, nutrition education, and increasing use of antenatal care, or by ensuring safe delivery. On the other hand, socio-economic and programmatic factors that focused on reducing post-neonatal and child deaths become more important during the post-neonatal period, when deaths are more often caused by infectious diseases or accidents. For example, it is obvious that immunization of the children or oral rehydration therapy will not prevent deaths during the neonatal period. It is, therefore, more useful to work with separate models for the determinants of competing risks of neonatal and post-neonatal death. In this study, we only consider the neonatal period.

In many countries, information on causes of death is not available. Verbal autopsy (VA) is a tool used in a retrospective interview with family members about the circumstances of a death to ascertain the underlying cause of death (Chen et al. 1980; Bhatia 1989; Baqui et al 2001; Karar et al. 2009). The interview is usually held 22 days after the date of death with the mother, or a close relative or neighbour in absence of the mothers (Karar et al. 2009). VA is not often used because it can be prohibitively expensive or difficult. The Health and Demographic Surveillance System (HDSS) of ICDDR,B in Matlab, Bangladesh, however, routinely records all births, deaths and causes of deaths through VA for a total population of about 220,000 (ICDDR,B 2006). It also incorporates information on several indicators of socioeconomic status of each household. The Matlab HDSS plays an important role in providing accurate information on vital events (e.g. births, deaths) and causes of death that are not often available in many resource-constrained setups.

In 1977, ICDDR,B started to provide extensive maternal-child health and family planning (MCH/FP) services, in addition to existing government health services, in half of the HDSS area called ICDDR,B area. The other half, called the comparison area, continued to get only the standard government health services. The MCH/FP project includes provision of domiciliary family planning services, simple nutrition education, tetanus toxoid immunization for pregnant women (which was modified in 1981 to include all women of reproductive age), community-based oral rehydration therapy, and measles immunization. These services were introduced incrementally phase by phase (Phillips et al 1984). In the ICDDR,B area there are several ICDDR,B sub-centres providing treatment for minor illnesses and, basic emergency obstetric care (EOC), and a permanent hospital that provides treatment for diarrhoeal diseases. In order to understand the way in which better health services shape child health, we analyze the data from the area with the better health care services in addition to the government health services (ICDDR,B area) as well as the data from an area with standard government health services only (the comparison area - a typical rural area of Bangladesh).

Most of the studies of cause-specific neonatal deaths in developing countries, including those in Matlab, Bangladesh, reported neonatal mortality rates by age-group, giving insight in when and why the child deaths occur (Chen et al. 1980; Bhatia 1989; Baqui et al. 2001; Lawn et al. 2006; Karar et al. 2009; Chowdhury et al. 2010). The trends in mortality, however, reveal that factors other than the health and family planning interventions influence the levels of mortality in the ICDDR,B and comparison areas (Bhatia 1989). To strengthen the targeting of interventions, it may therefore be important to analyze what characterizes families and children at risk of neonatal death due to various causes. In other words, what are the underlying factors that are associated with cause-specific deaths? Our study contributes to answering this question.

The analysis of cause-specific death is related to the concept of competing risks (Cornfield 1957; Fine and Gray 1999; Coviello and Boggess 2004). In a competing risk situation, the analysis of one specific cause of death has to account for competing causes of death. For example, in one study it was found that the probability of a female developing cancer at some point during her life has increased by 25 per cent over a seven years period, but the largest part of this increase was accounted for by decreases in other causes of mortality (Goldberg et al. 1956). To our knowledge, our study is the first that applies this type of model to neonatal mortality Bangladesh.

Duration models are often used in demographic and epidemiological research where the events to be modelled are associated with time, such as time till marriage, time from marriage till birth of the first child, or time till death (Cox 1959; Heckman et al. 1985). Analyzing the duration to competing causes of death leads to knowledge about when (time) and why (disease types) deaths occur, which is useful for targeting policies of early prevention.

Studies of child deaths have shown that survival chances of several children in a family are correlated and that variation in death risks across families remains after controlling for

observed covariates (such as age of mother at birth, gender of the child, or race), and can be attributed to unobserved family level heterogeneity. Examples are adverse genetic traits, inability to take care of the child (behavioural factors), or (unobserved) environmental factors (Mosley and Chen 1984; DasGupta 1990; Arulampalam and Bhalotra 2006).This is also relevant for specific causes of death. For example, if a mother has a propensity to give birth to children with too low birth weight, it may well happen that all births to this same mother expose too low weight at birth (genetic traits). Furthermore, closely spaced births of the same mother may be affected by infectious diseases of siblings (disease contamination) or environmental factors such as unsafe water supply or limited access to health care. Finally, as emphasized by DasGupta (1990), some mothers may be less resourceful in caring for their child than others, reflecting a behavioural effect.

5.3 Data

We combined the HDSS data of all live births and deaths of children for the ICDDR,B and comparison area obtained between 1987 and 2005. The data set has records of 107,367 singleton live births (57,830 from the comparison area and 49,837 from the ICDDR,B area) and 4,047 neonatal deaths and their causes (2,446 from the comparison area and 1,601 from the ICDDR,B area). Data on education of both mother and father, occupation of the father, and source of drinking water were obtained from the 1996 and 2005 census. For our purposes, we model the two areas separately.[16]

5.3.1 Causes of death

Since 1966 HDSS has recorded data on causes of death, with particular emphasis on child and maternal deaths. Before 1987, the cause of death was assigned by the health assistant, but from 1987 onwards the death form was revised and read by physicians who assigned a cause of death. A single three-digit code was selected from a list of 97 possible codes derived from the 'basic tabulation list' of the World Health Organization International Classification of Diseases, Injuries and Causes of Death (World Health Organization 1977). The death of a child often has more than one cause. The assignment of cause of death followed a hierarchical process whereby certain diagnoses were viewed as more certain than others, and thus given priority as primary and underlying cause of death. The assignment of causes of death is described details in the literature (see for example, Fauveau et al. 1994; Adjuik et al. 2006; Lawn et al. 2006; Karar et al. 2009; Ronsmans et al. 2010).

For our statistical analysis, to reduce the sampling error resulting from small numbers, the causes of neonatal deaths are first recoded into two categories (1) communicable diseases (CDs): acute respiratory infections, diarrhoea, dysentery, sepsis, meningitis, hepatitis, chicken pox, and

[16] Our primary interest of this research is to investigate the trends, pattern and determinants of epidemiological shifts in the ICDDR,B area and the comparison area. Some efficiency could possibly be gained by analyzing the two areas jointly, but this would also require at least some interactions of covariates with an area dummy (similar to one of the sensitivity checks in chapter 2). We tried this but encountered convergence problems.

neonatal tetanus or Extended Program on Immunization (EPI) related diseases and other viral diseases, (2) non-communicable diseases (NCDs): preterm delivery/low birth weight(LBW), deaths related to neonatal conditions (birth asphyxia, birth trauma/cord haemorrhage, congenital abnormality, neonatal infections, obstetric complications of new born, sudden infant death, unspecified neonatal death and miscellaneous neonatal deaths). Second, for an analysis at a more disaggregate level, exits due to non-communicable diseases (NCDs) of neonatal deaths were split into three categories: (1) LBW: preterm delivery/low birth weight, (2) NCs: deaths related to neonatal conditions (BA: birth asphyxia, CA: congenital abnormality or birth trauma/cord haemorrhage, NEO: neonatal related other conditions, OBSCOMP: obstetric complications), (3) Other (sudden infant death/unspecified or miscellaneous). See Table 1 in the annex for further details.

5.3.2 Socio-demographic variables

The covariates in the model refer to gender of the child, child birth cohorts (1987-1992, 1993-1999 or 2000-2005), religion of the family (Muslim or Hindu), dummies for birth order, education of the parents, employment status of the mother and occupation of the father (day labourer or not), source of drinking water (piped water or not), the mother's age at birth, and the distance to the nearest health facility. Maternal education is a proxy of child care skills and the ability to use modern health care services. Both paternal education and occupation are considered here as household socioeconomic indicators. The birth cohort of child can capture the time trends of cause-specific mortality risks. The distance variable captures the availability of health services, and sources of drinking water the environmental effects. Birth order variables are included to capture sibling effects on the risks of cause specific mortality. Summary statistics of these variables are presented in Table 1.

5.3.3 Distribution of causes of deaths

Table 2 depicts the percentage distribution of causes of neonatal death in the neonatal period (0-28 days). Of all neonatal deaths recorded in Matlab during 1987-2005, deaths due to NCDs comprised 87% and 78% in the ICDDR,B and comparison areas, respectively. The specific types of NCDs demonstrate that prematurity or low birth weight is a leading cause of death, followed by deaths 'unable to specify'. Among deaths due to CDs, the majority were due to acute respiratory infections (10.79 and 15.35 percent).

Figures 2 and 3 demonstrate that over the period 1987-2005, the fraction of neonatal deaths due to prematurity or low birth weight decreased, particularly in the ICDDR,B area. The fraction of deaths due to miscellaneous causes (sudden infant death, unable to specify, other disorders originated in the perinatal period etc.) also fell over time. Figures 4 and 5 show that deaths due to acute respiratory infections form an inreasing share of death due to communicable diseases in the comparison area, with neonatal tetanus or EPI related deaths disapprearing in both areas.

5.3.4 Cumulative incidence of cause-specific death

Before proceeding with advanced competing risks modelling we calculate the non-parametric cumulative incidence functions for the different causes of death to show how the cause-specific mortality changes with the age of the child. The value of the *cumulative incidence function* of cause *j* at time *t* is the probability of dying due to cause *j* before age *t*. The cumulative incidence is a function of the hazards of all the competing events and not solely of the hazard of the event to which it refers. See, for example, Coviello and Boggess (2004). Figures 6 and 7 show the cumulative incidence functions for neonatal deaths due to different causes based upon the complete samples in the ICDDR,B area and the comparison area, respectively.

The figures show that about 15 deaths per 1,000 live births accounted are ascribed to low birth weight (LBW) in both areas. About ten of these deaths occur in the first three days after birth. The second most frequent cause is unspecified or miscellaneous (OTHER), with about six and nine neonatal deaths per 1,000 births in the ICDDR,B and comparison area, respectively. These deaths are somewhat less concentrated in the first few days. This applies even more to deaths due to communicable diseases ARI and EPI, which together account for almost ten deaths per 1,000 births in the comparison area and five in the ICDDR,B area. Only about half of these are in the first week after birth. On the other hand, deaths due to obstetric complications (OBSCOMP) and birth asphyxia (BA) are almost exclusively concentrated in the first few days of life. Overall, although levels of neonatal deaths due to the various causes are substantially different in the two areas, the patterns of how these deaths are distributed over the 28 days of the neonatal period are similar in both areas.

5.4 Modelling

The modelling approach used builds on the concept of competing risks. We observe δ_{ij} =1 if child *i* dies from cause *j* (*j* =1,….,*k*) during the first 28 days after birth. We assume each neonatal death is associated with one single cause; there are in total *k* possible causes; we will estimate models with *k=2* and with *k=4*. The hazard of dying from cause *j* (*j*=1,….,*k*) at time *t* is denoted by $\lambda_j(t)$, where *t* refers to the age of child in days (0-28). In the competing risks model, we only observe the time till the first *of k* possible exits or until the end of the neonatal period, so that the observed survival time is given by $T_i = \min(T_{i1}, \ldots..,T_{ik}, C_i)$, where in our case C_i =28 days for each child *i*. So T_i is time of death in case of a neonatal death and 28 days in case of neonatal survival.

For the two exits model, $\lambda_1(t)$ is the hazard of dying due to CDs and $\lambda_2(t)$ the hazard of dying due to NCDs at time *t*. The hazard of dying at time *t,* given survival until *t,* due to any cause is given by $\lambda_1(t) + \lambda_2(t)$. In general, the hazard of dying at time *t* is given by $\lambda_1(t) + ... + \lambda_k(t)$. This sum corresponds to the single hazard $\lambda(t)$ of dying in the basic hazard model.

The hazard rates are specified as the following mixed proportional hazards (see for example Manton et al. 1981 or Lancaster 1979):

$$\lambda_j(t \mid x, v_j) = \lambda_{0j}(t)\exp(x\beta_j)v_j,\ j = 1,...,k \qquad (1)$$

The hazard rates are functions of time *t*, explanatory variables x which in our case do not vary over the neonatal period (child, mother and community level observed characteristics), and time constant mother-specific heterogeneity v_j. The explanatory variables *x* are assumed to enter through linear indexes $x\beta_j,\ j = 1,...,k.$ Time dependence is incorporated with a piecewise constant baseline hazard $\lambda_{0j}(t)$: for each cause of death *j* (*j*=1,…,*k*), we have $\lambda_{0j}(t)$ = $\exp(\beta_{0j})\sum_{h=1}^{H}\exp(\gamma_{hj})I_h(t)$ with $I_h(t) = I[t_{h-1} \le t < t_h]$, the indicator function for the interval $[t_{h-1}, t_h]$, and t_0 = 0, t_H = 28 days. Any duration dependence can be approximated arbitrarily closely by increasing the number of intervals. We experimented with several partitions of [0, 28] into several intervals and found the best model performance for the *H=5* intervals [0,1), [1,2), [2-3), [3,7) and [7,28]. For identification we need to restrict one of the γ_{hj} *(h=1,…,H)* to zero for each *j*. We choose $\gamma_{Hj} = 0$. Thus, β_{0j} determines the hazard in the last interval. The other γ_{hj} determine the ratio of the hazard in each interval compared to this last interval. The baseline hazard at $t \in [t_{h-1}, t_h)$ is higher than the baseline hazard for a duration of $t > t_h$ if $\gamma_h > 0$ and lower if $\gamma_h < 0$.

Our emphasis is on the specification of unobserved heterogeneity $v_j,\ j = 1,...k$, capturing unobserved factors that affect survival of a child. Ignoring these factors may bias the parameter estimates. In principle, unobserved heterogeneity can be child specific, mother specific, or community specific. Following several existing studies emphasizing the role of mother specific heterogeneity or 'frailty,' (Sastry 1997; Arulampalam and Bhalotra 2006) we model mother specific heterogeneity only. The unobserved heterogeneity terms $v_j > 0$ are time-independent and independent of observed characteristics *x*. Many different choices for the distribution of the unobserved heterogeneity exist. One issue is that the unobserved heterogeneity terms v_j of different causes of death *j* can be correlated. To address this we adopt a two factor loading model, with two independent fundamental factors W_1 and W_2 both having a discrete distribution on {-1,1}. This implies that

$$v_j = \exp(\alpha_{j1}W_1 + \alpha_{j2}W_2),\ j=1,...,k \qquad (2)$$

Let $W = (W_1, W_2)'$ and let *A* be the matrix of factor loadings with rows $A_j = (\alpha_{j1}, \alpha_{j2})$. Then the covariance matrix of the logarithms of the unobserved heterogeneity terms $v = (v_1,..., v_k)$ is given by V(ln(*v*)) = *A*V(*W*)*A*'. One additional restriction is needed for identification, we choose α_{12} =0. The probabilities for the discrete distributions for W_1, W_2 are Pr(W_1 = -1)= p_1 and

Pr(W_2 = -1)= p_2. We assume for both p_1 and p_2 a logit form, i.e. $p_1 = e^{\theta 1}/(1+ e^{\theta 1})$ and $p_2 = e^{\theta 2}/(1+ e^{\theta 2})$ and we estimate the θ's. Thus, for example, for our model with two exits (CDs and NCDs) the unobserved heterogeneity terms have the following distribution:

$$P(\ln v_1 = -\alpha_{11}, \ln v_2 = -\alpha_{21} - \alpha_{22}) = p_1 p_2;$$
$$P(\ln v_1 = -\alpha_{11}, \ln v_2 = -\alpha_{21} + \alpha_{22}) = p_1(1-p_2);$$
$$P(\ln v_1 = \alpha_{11}, \ln v_2 = \alpha_{21} - \alpha_{22}) = (1-p_1)p_2;$$
$$P(\ln v_1 = \alpha_{11}, \ln v_2 = \alpha_{21} + \alpha_{22}) = (1-p_1)(1-p_2).$$

The parameters can be estimated jointly with maximum likelihood in Stata; details on the likelihood function are available upon request. The covariance matrix of the unobserved heterogeneity terms can be estimated ex post, since it is a function of model parameters. This also applies to the *total survival* and *cumulative incidence functions.* The *total survival function* conditional on observed and unobserved heterogeneity is

$$S(t|X, v_1, \ldots, v_k) = \Pr(T \geq t|x, v_1, \ldots, v_k)$$
$$= \exp\left(-\sum_{j=1}^{k} v_j \int_0^t \lambda_{0j}(s) exp\left(x\,\beta_j\right) ds\right) \quad (3)$$

The *cumulative incidence function* of cause *j* is the probability of dying due to cause *j* before age *t*. In section 3, we have already presented the empirical cumulative incidence functions for various causes for the complete samples in the two areas. Based upon the model, we can also estimate the cumulative incidence functions for specific mothers, that is, conditional on observed and unobserved heterogeneity. They are given by:

$$F_j(t|x, v_1, \ldots, v_k) = \int_0^t v_j\, \lambda_{0j}(s) exp\left(x\,\beta_j\right) S(s|x, v_1, \ldots, v_k) ds \quad (4)$$

Integrating out the observed and unobserved heterogeneity, we can also obtain the total survival and cumulative incidence functions. Note that the sum of all cumulative incidence functions at a given age is equal to one minus the total survival function at that age, i.e.

$$\sum_{j=1}^{k} F_j(t|x, v_1, \ldots, v_k) = 1 - S(t|x, v_1, \ldots, v_k).$$

5.5 Estimation results

5.5.1 Communicable versus Non-communicable diseases: Covariate effects

Tables 3 and 4 present the estimation results for the models distinguishing two causes of death (non-communicable (NCDs) and communicable diseases (CDs)) in the ICDDR,B area and the comparison area, respectively. Tables 5 and 6 present the results for the same models with four causes of death, distinguishing CDs and three types of NCDs: low birth weight (LBW), neonatal related conditions (NCs), and unspecified/other (Other). We focus on the competing risk model introduced in the previous section, controlling for observed covariates in all the hazards and for mother specific unobserved heterogeneity terms that are allowed to be correlated across causes. For comparison, Tables 3 and 4 also present the parameter estimates of standard hazard models without unobserved heterogeneity for each cause of death separately ("traditional model"; first column in each table). In general, the estimated effects of the covariates are similar in the traditional model and in the full model, in terms of sign, size, and significance level. Allowing

for a general form of unobserved heterogeneity therefore has very little effect on the estimated duration dependence (the coefficients in the baseline intensity) or the estimated effects of the exogenous variables.

Tables 3 and 4 show that, in both areas, male children are more likely to die of CDs or NCDs than female children in similar families and circumstances. The gender differences are larger and more significant in the ICDDR,B area, and, in relative terms, larger for CDs than for NCDs. The magnitude of the differences is substantial. For example, in the ICDDR,B area, the chances of dying of a communicable disease on a given day in the neonatal period are about 70% higher (exp(0.54)-1)*100%) for a boy than for a girl (ceteris paribus). For a reference individual this is a difference of about 11 deaths per 1,000 over the complete neonatal period.

Religion, father's occupation and distance to the nearest health facility play no significant role in the ICDDR,B area. In the comparison area, however, a child is significantly more likely to die due to a CDs if the mother is Hindu (rather than Muslim), if the father is a day labourer, or if the distance to the nearest health facility is larger. The latter also applies to NCDs. It seems plausible that distance to the nearest health facility is more important in the comparison area than in the ICDDR,B area, since distances are much larger in the comparison area (cf. Table 1), making limited access to a health facility a more common concern there (Bhatia 1981).

A higher education level of the mother significantly reduces the NCDs hazard in both areas, whereas it has a negative but insignificant effect on death due to CDs. On average, if the mother is educated up at least secondary level this reduces the number of deaths per 1,000 live births due to NCDs on the first day after birth by 9 in the ICDDR,B area and by 14 in the comparison area.

In line with the demographic literature, first born children and children born to mothers aged less than 20 years old are at higher risk of neonatal mortality compared to the reference categories (20-24 years old mother and higher order births). The differences are much larger and more significant for NCDs than for CDs. In the ICDDR,B area, there is some evidence that children of older mothers (age 25 and older) have a lower risk of dying from CDs than the benchmark category (ages 20-24). There are no significant differences amongst birth orders 2 or higher.

A decreasing trend of neonatal death is observed in both CDs and NCDs in both areas, but it is not always significant. The degree of decline is strongest for CDs in the comparison area, where the risk has fallen substantially in the period 2000-2005. In the ICDDR,B area, there has been a significant reduction in the risk due to NCDs from the first to the second time period considered (a reduction of more than 25% from 1987-1992 to 1993-1999). In the comparison area, a similar reduction occurred a few years later.

A monotonically decreasing pattern trend is observed in the baseline intensity of dying due to NCDs in both areas: the hazard of dying is largest on the day of birth and already much

lower one day after birth, and decreases further during the neonatal period. On the other hand, the pattern is quite different for communicable diseases, for which the hazard declines much less during the first week (and even increases from day 0 to day 1). This difference is in line with Figures 6 and 7, where we already saw that deaths due to CDs less often occurred on the first few days after birth.

5.5.2 CDs and NCDs: Unobserved heterogeneity

The bottom panels of Tables 3 and 4 show that there is evidence of unobserved heterogeneity in both areas. In the ICDDR.B area, the covariance between the (mother specific) unobserved heterogeneity terms in the two competing hazards is significantly positive, implying a correlation coefficient of 0.28. In the comparison area the implied correlation is 0.66, but the estimated covariance is not significant. In both areas, only one of the variances is significantly different from zero, suggesting that it is hard to identify the covariance structure of the unobserved heterogeneity terms, possibly due to the fact that, fortunately, neonatal death due to each of the specific causes is not such a common event and more than one neonatal death in the same family is rare.

5.5.3 Model with four causes of death: Covariate effects

In order to get a better understanding of the deaths due to NCDs, we also split NCDs into three: (1) low birth weight (LBW); (2) neonatal conditions (NCs) which includes CA, NEO, BA, OBSCOMP; and (3) Other: sudden infant deaths/ unspecified or miscellaneous (miscellaneous: with a range of 27-30 cases in each area). Together with CDs, this gives four different causes of neonatal death. The results of these models for the complete model allowing for correlated unobserved heterogeneity in all four hazard rates are reported in Tables 5 and 6 for the ICDDR,B area and the comparison area, respectively. (Results of the corresponding traditional models generally give similar effects for the covariates; details are available upon request from the authors.) In general, the effects of many covariates are quite different for the three causes of death due to non-communicable diseases, showing that treating these causes separately is worthwhile.

The disadvantage for boys in NCDs that we already found in Tables 3 and 4 can be attributed to their larger vulnerability to NCs and, in the ICDDR,B area, to death due to low birth weight. We find no significant difference between boys and girls for the category “Other.” As in Table 3, no significant religion difference is found in the ICDDR,B area. In the comparison area, however, a child born to a Hindu mother is not only more likely than an otherwise similar child from a Muslim mother to die from a communicable disease, but is also more vulnerable to death related to NCs.

In both areas, higher education of the mother substantially reduces the risk of death due to LBW. The effects of mother’s education on the other causes of death are much weaker, though some are still significant at the 5% level. Children born to very young mothers (age less than 20

years) are more likely to die due to LBW and due to "other" causes in both areas. The effects of birth order show that first born children are at higher risk of death due to any cause than higher order births. The differences are large, sometimes more than a factor two; the only exception is death due to CDs in the ICDDR,B area where birth order appears to play no role.

Some of the results for the father's education level and type of occupation seem puzzling at first sight. In particular, we find that children of fathers with primary education rather than no education have higher risk to die because of NCs in both areas, and the effect is significant at the 5% level (but not at 1%). Moreover, if the father is a day labourer, this reduces the risk to die from NCDs in the ICDDR,B area, whereas being a day labourer is a negative index of socio-economic status. The finding that death due to NCs is positively associated with socio-economics status is in line, however, with data from the nationally representative BDHS 2004 (NIPORT et al. 2005), which show that deaths due to birth asphyxia are more common amongst mothers with higher education. An existing study (Chowdhury et al. 2010) suggests this may be related to delivery at a health centre instead of at home, and this finding is in line with large unobserved heterogeneity for deaths due to NCs and increased institutional delivery (which includes mainly birth asphyxia/neonatal infections and delivery complications) in the ICDDR,B area.[17] On the other hand, secondary education of the father reduces the risk to die from "other" causes in the comparison area, and in the same area, being a day labourer increases the risk of death due to CDs (as in Table 4), as expected.

Lack of access to running water has the expected effect of increasing mortality due to CDs in the ICDDR,B area. It also increases the risk of dying from LBW. On the other hand, it has no significant effects in the comparison area. In the comparison area, a larger distance to the nearest health centre increases the risk of CDs related death (as in Table 4) and of LBW related death. As in Table 3, it has no significant effect in the ICDDR,B area where distances are smaller. In both areas, we find substantial differences among cohort effects for different causes of death. In particular, the risk of NCs related death has increased in 2000-2005 compared to 1987-1999, while the risk of dying from the other causes has fallen. Particularly for the "other" category (which includes sudden infant deaths, among others) the reduction in the period 2000-2005 is remarkably large in both areas.

The baseline intensities of dying due to LBW, NCs, and other non-communicable diseases follow similar patterns, which are also similar to the patterns in Tables 3 and 4 for all NCDs combined: the risk is very high on the first day of life, and reduces quickly after a few days. The pattern is quite different from that for non-communicable diseases, for which the hazard shows a much less clear duration dependence pattern over the neonatal period, as we already saw in Tables 3 and 4.

[17] A study in Matlab revealed that 19% of births took place in ICDDR,B facilities, 4% occurred in other (public and private) facilities, 2.6 births were attended by ICDDR,B midwives at home, and the remaining deliveries took place at home (Anwar et al. 2004). However, according to BDHS 2004, nationally 90% of all births took place at home, which is comparable with our comparison area, a typical rural area of Bangladesh with the usual standard health facilities.

5.5.4 Model with four causes of death: Unobserved heterogeneity

We find some evidence of unobserved heterogeneity: two of the four variances are significant in the ICDDR,B area and one in the comparison area. Moreover, in the ICDDR,B area, the covariance between the unobserved heterogeneity terms in the hazards for communicable diseases and low birth weight related deaths is significant at the 1% level. The other covariances are insignificant at the 5% level. Still, at least in the ICDDR.B area, the covariance matrix of the unobserved heterogeneity terms seems easier to estimate in this model than in the model with only two causes of death – we no longer find the very large standard error found for NCDs in Table 3. This suggests that this large standard error might be due to aggregation of rather different causes of death. (That this problem does not arise in the comparison area may be because of the larger death rates there.)

5.5.5 Cumulative incidences functions

The cumulative incidence functions corresponding to the models with four causes of death for the benchmark cases are shown in Figures 8 (ICDDR,B area) and 9 (comparison area). These rates are substantially different from the rates for the complete sample (see Figures 6 and 7), since the socio-economic characteristics of the benchmark case are not representative for the sample average. The patterns over time confirm what we concluded from the baseline hazards in Tables 5 and 6: they are much steeper in the first few days for the various types of non-communicable diseases than for communicable diseases. At each point of time, the cumulative number of deaths due to LBW is larger than the numbers for all three other causes. In the ICDDR,B area, about 16 deaths per 1,000 live births are due to LBW within one day after birth, rising to about 28 per 1,000 after 28 days. The patterns over time are similar in the two areas, but the levels are not: the hazards for CDs and for “other” NCDs are much larger in the comparison area than in the ICDDR,B area.

Figures 10 and 11 show the same cumulative incidence functions as Figures 8 and 9, but now for a benchmark birth in the period 2000-2005 instead of 1987-1992. The epidemiological shifts are similar in the two areas. In both areas, the largest difference between the two time periods is the significant reduction of the number of deaths due to LBW, about 12 per 1,000 in the ICDDR,B area and 8 per 1,000 in the comparison area during the whole neonatal period, and concentrated in the first few days after birth. On the other hand, the number of deaths due to NCs surprisingly increases substantially in the period 2000-2005, by about 5-6 deaths per 1,000 live births compared to the reference period 1987-1992. In the competing risks situation, the decrease of deaths due to LBW is perhaps partly substituted by an increase of deaths due to neonatal infections or obstetric complications at birth.

5.6 Discussion and conclusion

This study analyzes causes of neonatal death, derived from open-ended death history data reported by the mother or a close relative or neighbour (in absence of the mother) and recorded by non-medically trained field workers. Three physicians independently reviewed all death records and reached consensus. The uniform death registration form and assessment of causes of death by physicians during 1987-2005 is an important strength for the comparison the patterns of causes of death over the years. This Verbal Autopsy method was recommended by WHO to attain the reliable epidemiological data on mortality (Fauveau at el. 1994; Bryce et al. 2005).

During 1987-2005, recorded neonatal mortality per 1,000 live births was 32.3 in the ICDDR,B area (which, in addition to government services, gets high quality health care services) and 42.3 in the comparison area (with standard government services). In Bangladesh, the national neonatal mortality rate is about 41 per 1,000 live births (BDHS, 2004) and this rate is close to the rate of comparison area, a typical setup in rural Bangladesh.

A remarkable success is the reduction in the number of neonatal deaths due to neonatal tetanus or EPI, which mainly explains the reduction of total neonatal mortality in ICDDR,B area, and which is well noticed in other studies (Bhatia 1989; Baqui et al. 2001). This is supported by the cause-of-death data, indicating that mortality rates due to neonatal tetanus were 1.0% and 5.9% per 1,000 live births in ICDDR,B and comparison areas, respectively. During the study period, low birth weight was the foremost leading cause of neonatal deaths in both ICDDR,B and comparison area. Our findings are in agreement with global findings pointing at preterm birth or low birth weight as a major cause of neonatal death in the world and particularly in Bangladesh (Lawn et al. 2006).

On the other hand, death related to NCs - mainly neonatal infections or obstetric complications - became a primary cause of neonatal death in both areas in 2000-2005 (see also, Chowdhury et al., 2010). Compared to the 1987-1992 period, for otherwise similar children, the hazard to die due to NCs increased by about 77% in the ICDDR,B area and 46% in the comparison area. The increase in this rate in the ICDDR,B area is remarkable. As indicated in an earlier study, the absence of appropriate antenatal, intra-partum, and postnatal care in both areas takes an unnecessary toll on infant lives, which could easily be prevented with appropriate interventions (Bhatia 1989; Bari et al. 2002; Bang et al. 2005; Velaphi and Pattinson 2007).

Although a downward trend since 1993 is observed in neonatal death due to NCDs in both areas, this decline is faster in the ICDDR,B area, specifically for deaths due to LBW. This finding can be related to the large scale nutrition programs in the ICDDR,B area, which attempt to improve nutrition of pregnant mothers with the goal of increasing birth weight (see www.icddrborg/what-we-do/health-programmes/nutrition). Since villages in the comparison area are contiguous to those in the ICDDR,B area, spill-over effects of these programs, changing information and behaviour in the comparison area also, may explain why mortality due to LBW has also significantly declined in the comparison area (Phillips et al. 1988).

The decline in childhood deaths due to CDs is widely discussed in the epidemiology literature. It is mainly due to neonatal tetanus or EPI, which is no longer an existing cause of death after 2000 in either area. Existing studies for India (Reddiah and Kapoor 1988) and Bangladesh (Bhatia 1989) show that the numbers of deaths due to CDs (including acute respiratory infections) remained almost unchanged over the period 1993-2005. In contrast, a recent study in Bangladesh found a reduction of 79% in child or infant deaths due to respiratory infections during 1986-2006 (Karar et al. 2009). It may be noteworthy to mention that lack of consistent case definitions and rules in the hierarchical assignment of causes may hinder comparisons across time and studies.

The time of exposure to a disease (the number of days after birth) is an important phenomenon in epidemiological studies. This study finds that the number of children dying during days 1-6 due to all types of NCDs falls over the period, but on the other hand the number of deaths due to CDs increases. Studies in Matlab (Bhatia 1989; Chowdhury et al. 2010) reported that the ICDDR,B program significantly reduced neonatal mortality within one day after birth, which is also apparent in our estimations (Figures 10 and 11).

Our result confirms the general conclusion of the levels, trend and pattern of causes of neonatal deaths, but we find some remarkable socioeconomic differences in the cause-specific deaths. Cause-specific deaths due to low birth weight and other causes (sudden deaths, specified, unspecified) are better explained from the socio-economic covariates than the others. Secondary education of the mother reduces deaths due to LBW significantly and thus it seems that education helps women to improve general socioeconomic status or overcome the barriers set by low autonomy in traditional society. Education improves women's innate ability in pregnancy management and in caring for their child and management of household work (DasGupta 1990).

First-borns and children born to a young mother (age below 20) are more likely to die due to LBW in both areas, but particularly in the comparison area. This reflects an advantage of high quality primary care services and interventions for the risk of low birth weight (LBW) in the ICDDR,B area. Father's education leads to lower risk of neonatal mortality due to LBW in the ICDDR,B area, possibly as an indicator of the family's general socioeconomic status, which helps to take advantage of high quality services in ICDDR,B area. In the comparison area on the other hand, the father's being a day labourer, another index of poor socio-economic status, makes neonatal death due to CDs more likely.

Neonatal death is more likely among Hindu families (due to CDs and NCs) in the comparison area and a similar trend is observed in a study for India (Bhalotra et al. 2010a,b). In the ICDDR,B area, extensive health services apparently annihilate this religion difference. Male children are more likely to die than female children due to CDs and NCDs in both areas. The (relative) difference is largest for CDs in the ICDDR,B area (almost 72%). Furthermore, gender discrepancies in deaths due to NCDs are mainly related to NCs. This is in line with a study that

finds that infant mortality is inherently larger for boys than for girls, but that this can be reversed by environmental disadvantages for female children. (Waldron 1983; Chowdhury et al. 2010). The influence of such environmental factors can be reduced by the extensive health services in the ICDDR,B area. This finding gives an insight of what causes gender discrepancies in child deaths compared to an earlier study which only revealed overall improvement of female child survival in the ICDDR,B area (Datta and Bairagi 2000). No significant gender difference is observed for neonatal deaths due to LBW in the ICDDR,B area, perhaps since nutrition programs in ICDDR,B area diminished the excess deaths due to malnutrition among female children, where earlier studies reported excess female deaths (Bhuiya et al. 1986; Fauveau et al. 1991).

Keeping constant socioeconomic indicators (parental education and occupation) in the model, the risks for first-borns and children of young mothers remain significantly higher than for others, probably pointing at a role of physiological factors rather than socio-economic factors (Bhatia 1989).

An additional contribution of our study is to allow for a flexible form of unobserved heterogeneity. We could not include some potentially relevant covariates, such as use of antenatal care and birth practice, which might lead to unobserved variation in the outcomes of our interest. Unobserved heterogeneity in death due to LBW may reflect the importance of extra attention to warmth, feeding and prevention or early treatment of infections (Conde-Agudelo et al. 2003). Point estimates of large unobserved heterogeneity in the hazards of NCs (mainly neonatal related infections or obstetric complications) suggest disadvantages or mistreatment of modern health technology or lack in quality of care. For example, unnecessary administration of oxytocics to augment labour in child birth or inadequate foetal monitoring by health workers increased neonatal deaths significantly (Bari et al. 2002; Bang et al. 2005;Velaphi and Pattinson 2007).

Our findings highlight the role of maternal and child health interventions for child survival, particularly tetanus toxoid immunization for pregnant women, nutrition programs, and high coverage health services (distance to health centre and information dissemination). Death due to EPI has been eliminated, but in order to achieve MDG-4 of reducing child mortality, strategies targeting acute respiratory diseases remain necessary.

For further reduction of neonatal mortality due to low birth weight it is important to add strategies to ensure equitable utilization of services by various socio-economic groups to the existing programs, such as for low educated mothers, and particularly for their first pregnancy. On the other hand, the finding that unobserved heterogeneity in the ICDDR,B area is much larger for deaths due to NCs than in the comparison area suggests more death tolls because of poor quality of institutional delivery and in foetus monitoring. It also may mean that not everyone benefits equally from the health interventions, so that policies that increase quality and equity in interventions may help to further reduce neonatal mortality.

Tables

Table 1. Summary statistics of explanatory variables.

Variable	ICDDR,B area	Comparison area
Gender of index child		
Male	50.78	50.76
Child's birth cohort		
Before 1993	32.86	36.53
1993-1999	34.63	34.26
2000-2005	32.51	29.22
Birth order		
1	30.97	26.65
2-3	43.07	38.23
4 +	25.96	35.12
Religion: Hindu	14.67	8.91
Mother's education		
No education	49.00	53.10
Primary education	25.15	25.05
At least secondary education	25.86	21.85
Mother's age at birth		
<20	12.19	11.54
20-24	33.31	33.13
25-29	28.55	28.39
30 +	25.94	17.8
Father's education		
No education	57.26	58.72
Primary education	21.70	23.00
At least secondary education	21.04	18.28
Father's occupation		
Day labourer	15.75	18.37
Source of drinking water		
No tube-well/pipewater	25.31	24.10
Distance to health centre	1.86 (0.96)	7.29 (3.96)

Note: Percentages of outcome 1 for all dummy variables; mean and standard deviation (in parentheses) for continuous variables.

Table 2. Percentage distribution of cause-specific neonatal deaths.

Cause of death	ICDDR,B area	Comparison area
	Neonatal deaths (0-28 days)	Neonatal deaths (0-28 days)
Communicable diseases (CDs)	**13.09**	**21.93**
Hepatitis	0.06	0.00
Septecaemia	0.31	0.00
Acute respiratory infections (ARI)	10.79	15.35
Diarrheal diseases	0.93	0.73
Neonatal tetanus or EPI related (EPI)	1.00	5.85
Non-communicable diseases (NCDs)	**86.57**	**77.90**
Congenital abnormality	2.43	1.71
Prematurity/low birth weight (LBW)	45.95	36.50
Birth asphyxia (BA)	5.25	3.68
Obstetric complications of new born (OBSCOMP)	8.74	9.20
Birth trauma/cord haemorrhage	1.69	1.02
Other neonatal related conditions* (NEO)	4.10	4.15
Miscellaneous**	2.05	1.17
Diagnosis not possible***	16.36	20.47

* includes neonatal infections, respiratory and cardiovascular specific disorder to the perinatal period

**includes skin infections, fever, jaundice, intestinal obstruction, Oedemas, external cause (injury), homicide

*** includes sudden infant death, unspecified cause, other disorder in the perinatal period

Table 3. Parameter estimates of competing risks model for neonatal deaths due to communicable and non-communicable diseases, ICDDR,B area.

Variable	Communicable Diseases (CDs)				Non-communicable Diseases (NCDs)			
	Traditional		With Unobserved Heterogeneity		Traditional		With Unobs. Heterogeneity	
Male	0.53**	(0.14)	0.54**	(0.14)	0.21**	(0.05)	0.23**	(0.06)
Hindu	-0.02	(0.20)	-0.03	(0.20)	0.01	(0.08)	0.001	(0.08)
Mother's education level								
At least primary	-0.17	(0.18)	-0.19	(0.18)	-0.13*	(0.07)	-0.15*	(0.07)
At least secondary	-0.19	(0.23)	-0.23	(0.22)	-0.35**	0.09)	-0.39**	(0.09)
Mother's age at birth								
<20 years	0.30	(0.21)	0.29	(0.22)	0.20*	(0.08)	0.20*	(0.08)
25-29 years	-0.50*	(0.23)	-0.47*	(0.21)	-0.08	(0.09)	-0.05	(0.09)
≥30	-0.58*	(0.26)	-0.52*	(0.26)	0.13	(0.10)	0.20*	(0.10)
Birth order								
2-3	-0.02	(0.20)	-0.08	(0.20)	-0.67**	(0.08)	-0.73**	(0.08)
≥4	0.26	(0.28)	0.13	(0.29)	-0.51**	(0.11)	-0.65**	(0.11)
Father's education level								
At least primary	-0.17	(0.18)	-0.17	(0.18)	0.17*	(0.07)	0.17*	(0.07)
At least secondary	-0.30	(0.21)	-0.30	(0.22)	-0.08	(0.08)	-0.07	(0.09)
Father day labourer	0.20	(0.18)	0.18	(0.18)	-0.02	(0.08)	-0.03	(0.08)
No tube-well/pipe water	0.31*	(0.15)	0.31*	(0.16)	0.12*	(0.06)	0.11	(0.07)
Distance to health centre [d]	0.01	(0.07)	0.01	(0.07)	-0.01	(0.03)	-0.005	(0.03)
Birth cohort child								
1993-1999	0.08	(0.16)	0.05	(0.16)	-0.29**	(0.07)	-0.31**	(0.07)
2000-2005	-0.30	(0.21)	-0.32	(0.20)	-0.31**	(0.07)	-0.33**	(0.08)
Baseline intensity								
Day 0	0.80*	(0.29)	0.76*	(0.29)	4.11**	(0.07)	4.06**	(0.07)
Day 1	1.28**	(0.24)	1.26**	(0.24)	2.55**	(0.10)	2.52**	(0.10)
Days 2	1.02**	(0.27)	1.00**	(0.27)	2.01**	(0.12)	1.99**	(0.12)
Days 3-6	0.55**	(0.18)	0.54**	(0.18)	1.15**	(0.10)	1.14**	(0.10)
Constant	-9.07**	(0.21)	-7.97**	(0.37)	-7.83	(0.11)	-7.59**	(2.19)
Unobserved heterogeneity								
Variance	-	-	0.14*	(0.07)	-	-	1.58	(3.01)
Covariance	-	-	0.13*	(0.06)	-	-	-	-

Notes: [d] centered around its mean in each area; * p-value<0.05, ** p-value<0.01, standard errors are in parentheses

Reference category: gender is female, religion is Muslim, mother and father have no education, mother's age at birth 20-24 years, father is not day-labourer, source of drinking water is tube-well/pipewater, living at average distance to health centre, child birth cohort 1987-1992, baseline intensity 7-28 days.

Table 4. Parameter estimates of competing risks model for neonatal deaths due to communicable and non-communicable diseases, comparison area.

Variables	Communicable Diseases (CDs)				Non-communicable Diseases (NCDs)			
	Traditional		With Unobs. Heterogeneity		Traditional		With Unobserved Heterogeneity	
Male	0.19*	(0.09)	0.20*	(0.09)	0.11*	(0.05)	0.11*	(0.05)
Hindu	0.28*	(0.14)	0.28*	(0.14)	0.13	(0.08)	0.14	(0.08)
Mother's education level								
At least primary	-0.21	(0.12)	-0.22	(0.12)	-0.10	(0.06)	-0.11	(0.06)
At least secondary	-0.23	(0.15)	-0.25	(0.15)	-0.35**	(0.08)	-0.37**	(0.08)
Mother's age at birth								
<20 years	0.12	(0.14)	0.12	(0.14)	0.27**	(0.07)	0.26**	(0.07)
25-29 years	-0.27	(0.13)	-0.25	(0.13)	-0.03	(0.07)	0.001	(0.07)
30 years plus	-0.14	(0.17)	-0.10	(0.16)	0.05	(0.09)	0.11	(0.09)
Birth order								
2-3	-0.33**	(0.12)	-0.38**	(0.13)	-0.63**	(0.67)	-0.68**	(0.07)
4 plus	-0.20	(0.17)	-0.29	(0.17)	-0.61**	(0.09)	-0.71**	(0.09)
Father's education level								
At least primary	0.01	(0.11)	0.014	(0.11)	0.04	(0.06)	0.06	(0.06)
At least secondary	-0.20	(0.15)	-0.20	(0.14)	-0.03	(0.07)	0.03	(0.07)
Father day labourer	0.46**	(0.10)	0.48**	(0.10	0.12*	(0.06)	0.13*	(0.06)
No tube-well/pipe	0.03	(0.11)	0.03	(0.11)	0.04	(0.06)	0.04	(0.06)
Water								
Distance to health	0.03*	(0.01)	0.03*	(0.01)	0.02**	(0.01)	0.02**	(0.01)
Centre [d]								
Birth cohort child								
1993-1999	-0.20	(0.11)	-0.21	(0.11)	-0.10	(0.06)	-0.11	(0.06)
2000-2005	-0.49**	(0.13)	-0.50**	(0.13)	-0.28**	(0.07)	-0.29**	(0.07)
Baseline intensity								
Day 0	1.02**	(0.16)	0.99**	(0.16)	3.96**	(0.06)	3.92**	(0.06)
Day 1	0.61**	(0.20)	0.58**	(0.20)	2.54**	(0.08)	2.51**	(0.08)
Days 2	0.37	(0.22)	0.35	(0.22)	1.90**	(0.11)	1.88**	(0.11)
Days 3-6	0.77**	(0.10)	0.76**	(0.10)	1.18**	(0.08)	1.17**	(0.08)
Constant	-8.05**	(0.16)	-7.57**	(0.58)	-7.69**	(0.10)	-6.80**	(0.33)
Unobserved heterogeneity								
Variance	-	-	0.41	(0.44)	-	-	0.38*	(0.13)
Covariance	-	-	0.26	(0.23)	-	-	-	-

Notes: [d] centered around its mean in each area; * p-value<0.05, ** p-value<0.01, standard errors are in parentheses

Reference category: gender is female, religion is Muslim, mother and father have no education, mother's age at birth 20-24 years, father is not day-labourer, source of drinking water is tube-well/pipewater, living at average distance to health centre, child birth cohort 1987-1992, baseline intensity 7-28 days.

Table 5. Parameter estimates of intensity to neonatal deaths due to communicable diseases and different types of non-communicable diseases, ICDDR,B area.

Variables	Communicable diseases (CDs)		Non-communicable Diseases (NCDs)					
	CDs		LBW[a]		NCs[b]		Other[c]	
Male	0.54**	(0.14)	0.26**	(0.08)	0.30**	(0.11)	0.07	(0.12)
Hindu	-0.03	(0.20)	-0.15	(0.12)	0.16	(0.15)	0.15	(0.16)
Mother's education level								
At least primary	-0.19	(0.18)	-0.07	(0.10)	-0.12	(0.14)	-0.38*	(0.16)
At least secondary	-0.23	(0.22)	-0.44**	(0.13)	-0.34*	(0.16)	-0.36	(0.19)
Mother's age at birth								
<20 years	0.29	(0.22)	0.24*	(0.11)	-0.04	(0.16)	0.38*	(0.18)
25-29 years	-0.47*	(0.21)	-0.21	(0.12)	0.09	(0.16)	0.16	(0.18)
30 years plus	-0.52*	(0.26)	0.16	(0.15)	0.30	(0.20)	0.23	(0.22)
Birth order								
2-3	-0.08	(0.20)	-0.67**	(0.11)	-0.96**	(0.15)	-0.57**	(0.17)
4 plus	0.13	(0.29)	-0.67**	(0.16)	-0.77**	(0.22)	-0.41	(0.24)
Father's education level								
At least primary	-0.17	(0.18)	0.12	(0.10)	0.33*	(0.13)	0.14	(0.15)
At least secondary	-0.30	(0.22)	-0.25*	(0.12)	0.11	(0.15)	0.08	(0.18)
Father day labourer	0.18	(0.18)	0.01	(0.10)	-0.51*	(0.18)	0.24	(0.15)
No tube-well/pipe water	0.31*	(0.16)	0.18*	(0.09)	-0.21	(0.14)	0.24	(0.13)
Distance to health centre[d]	0.01	(0.07)	-0.02	(0.04)	-0.04	(0.06)	0.08	(0.06)
Birth cohort child								
1993-1999	0.05	(0.16)	-0.36**	(0.09)	-0.24	(0.15)	-0.24	(0.14)
2000-2005	-0.32	(0.20)	-0.76**	(0.11)	0.57**	(0.14)	-0.66**	(0.17)
Baseline intensity								
Day 0	0.76*	(0.29)	4.09**	(0.10)	4.53**	(0.16)	3.46**	(0.15)
Day 1	1.26**	(0.24)	2.45**	(0.15)	2.93**	(0.21)	2.28**	(0.20)
Days 2	1.00**	(0.27)	2.08**	(0.17)	1.77**	(0.30)	1.97**	(0.23)
Days 3-6	0.54**	(0.18)	1.28**	(0.14)	0.80**	(0.26)	1.05**	(0.19)
Constant	-7.97**	(0.37)	-7.19**	(0.45)	-10.62**	(3.13)	-8.31**	(0.39)
Variance	0.14*	(0.07)	0.58*	(0.24)	3.42	(10.3)	0.23	(0.20)
Covariance (row1)	-	-	0.17**	(0.06)	0.13	(0.07)	0.16*	(0.07)
Covariance (row2)	-	-	-	-	-0.97	(1.63)	0.05	(0.28)
Covariance (row3)	-	-	-	-	-	-	0.55	(1.15)

Notes: [d] centered around its mean in each area; * p-value<0.05, ** p-value<0.01, standard errors are in parentheses

Reference category: gender is female, religion is Muslim, mother and father have no education, mother's age at birth 20-24 years, father is not day-labourer, source of drinking water is tube-well/pipewater, living at average distance to health centre, child birth cohort 1987-1992, baseline intensity 7-28 days.

[a] Low birth weight/prematurity

[b] Neonatal infections, birth asphyxia, obstetric complications, respiratory disorders, birth trauma, cord haemorrhage congenital abnormalities

[c] skin infections, fever, jaundice, intestinal obstruction, Oedemas, external cause (injury), homicide, and sudden infant death, unspecified cause, other disorder in the perinatal period

Table 6. Parameter estimates of intensity to neonatal deaths due to communicable diseases and different types of non-communicable diseases, comparison area.

Variables	Communicable diseases (CDs)		Non-communicable Diseases (NCDs)					
	CDs		LBW[a]		NCs[b]		Other[c]	
Male	0.19*	(0.09)	0.08	(0.07)	0.31**	(0.09)	-0.01	(0.09)
Hindu	0.28*	(0.14)	0.03	(0.12)	0.37*	(0.15)	0.08	(0.15)
Mother's education level								
At least primary	-0.21	(0.12)	-0.15	(0.09)	-0.03	(0.12)	-0.07	(0.11)
At least secondary	-0.23	(0.15)	-0.61**	(0.12)	-0.03	(0.14)	-0.36*	(0.15)
Mother's age at birth								
<20 years	0.12	(0.14)	0.36**	(0.10)	-0.09	(0.14)	0.44**	(0.14)
25-29 years	-0.27	(0.13)	-0.10	(0.11)	0.11	(0.14)	0.09	(0.13)
30 years plus	-0.14	(0.17)	0.09	(0.13)	0.14	(0.17)	0.14	(0.16)
Birth order								
2-3	-0.33**	(0.12)	-0.60**	(0.10)	-1.00**	(0.13)	-0.49**	(0.13)
4 plus	-0.20	(0.17)	-0.79**	(0.14)	-0.88**	(0.18)	-0.39*	(0.17)
Father's education level								
At least primary	0.004	(0.11)	0.10	(0.09)	0.23*	(0.11)	-0.16	(0.11)
At least secondary	-0.20	(0.15)	0.03	(0.11)	0.15	(0.13)	-0.32*	(0.15)
Father day labourer	0.46**	(0.10)	0.11	(0.09)	0.14	(0.12)	0.14	(0.11)
No tube-well/pipe water	0.03	(0.11)	0.17	(0.09)	0.024	(0.13)	-0.15	(0.11)
Distance to health centre[d]	0.03*	(0.01)	0.03**	(0.01)	0.02	(0.01)	-0.002	(0.01)
Birth cohort child								
1993-1999	-0.20	(0.11)	-0.04	(0.09)	-0.19	(0.13)	-0.15	(0.11)
2000-2005	-0.49**	(0.13)	-0.50**	(0.11)	0.38**	(0.13)	-0.78**	(0.14)
Baseline intensity								
Day 0	0.99**	(0.16)	3.93**	(0.09)	4.58**	(0.14)	3.37**	(0.11)
Day 1	0.58**	(0.20)	2.53**	(0.12)	2.81**	(0.19)	2.32**	(0.15)
Days 2	0.35	(0.22)	1.89**	(0.15)	1.62**	(0.29)	1.97**	(0.17)
Days 3-6	0.76**	(0.10)	1.15**	(0.12)	1.52**	(0.19)	1.01**	(0.14)
Constant	-8.05**	(0.16)	-7.34**	(0.40)	-9.20**	(0.43)	-7.79**	(0.44)
Variance	0.35	(0.29)	0.52*	(0.23)	0.16	(0.12)	0.26	(0.19)
Covariance (row1)	-	-	0.36	(0.24)	0.02	(0.13)	0.01	(0.11)
Covariance (row2)	-	-	-	-	0.18	(0.14)	0.21	(0.17)
Covariance (row3)	-	-	-	-	-	-	0.20	(0.14)

Notes: [d] centered around its mean in each area; * p-value<0.05, ** p-value<0.01, standard errors are in parentheses

Reference category: gender is female, religion is Muslim, mother and father have no education, mother's age at birth 20-24 years, father is not day-labourer, source of drinking water is tube-well/pipewater, living at average distance to health centre, child birth cohort 1987-1992, baseline intensity 7-28 days.

[a] Low birth weight/prematurity

[b] Neonatal infections, birth asphyxia, obstetric complications, respiratory disorders, birth trauma, cord haemorrhage congenital abnormalities

[c] skin infections, fever, jaundice, intestinal obstruction, Oedemas, external cause (injury), homicide, and sudden infant death, unspecified cause, other disorder in the perinatal period

Figures

Figure 1: Child mortality per 1000 live births, Source: BDHS 2007

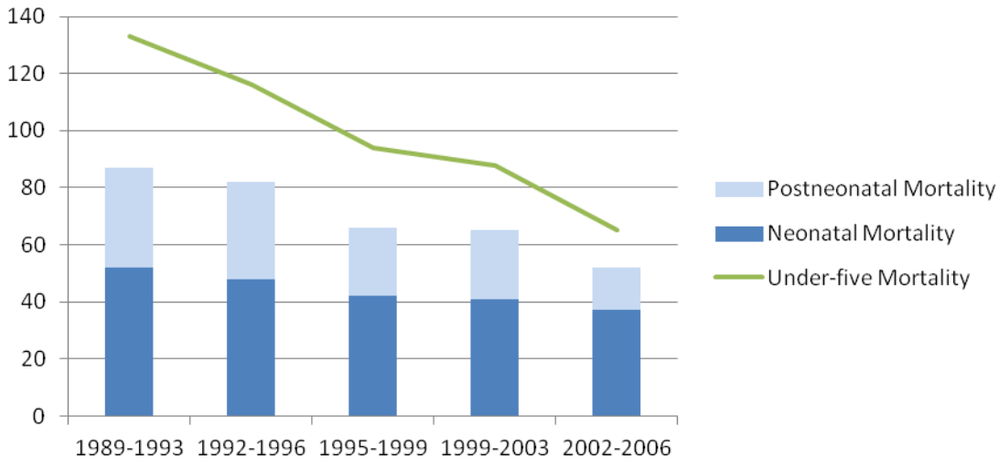


Figure 2: Neonatal deaths due to major non-communicable diseases, ICDDR,B area

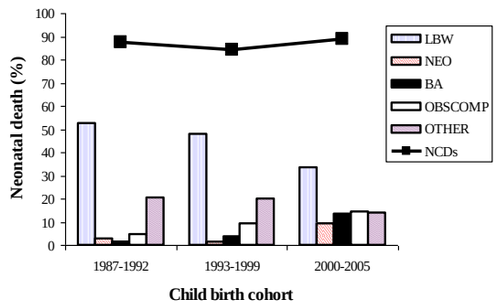


Figure 3: Neonatal deaths due to major non-communicable diseases, comparison area

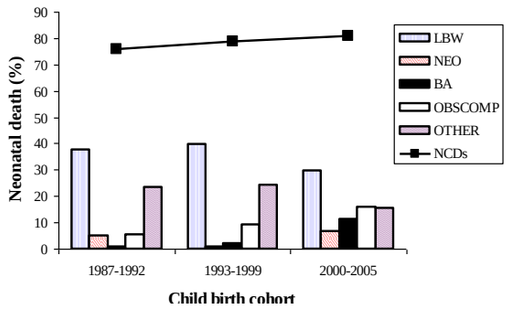


Figure 4: Neonatal deaths due to major communicable diseases, ICDDR,B area

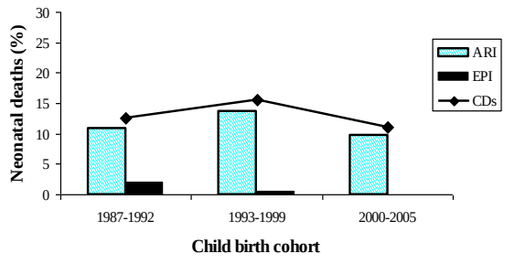
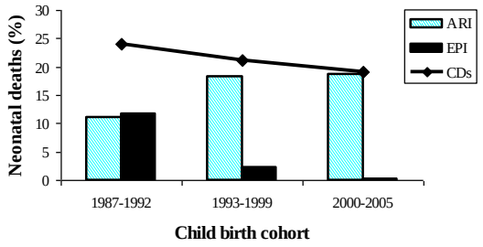


Figure 5: Neonatal deaths due to major communicable diseases, comparison area



Notes: NCDs: non-communicable diseases, LBW: low birth weight, NEO: neonatal related other conditions (infections, respiratory and cardiovascular disorder specific to the perinatal period), BA: birth asphyxia, OBSCOMP: obstetric complications, OTHER: sudden infant death, unspecified, other disorders originated in the perinatal period etc. CDs: communicable diseases, ARI: acute respiratory infections/pneumonia, EPI: extended program for immunization related diseases.

Figure 6: Nonparametric estimates of cause-specific neonatal death
ICDDR,B area, 1987-2005

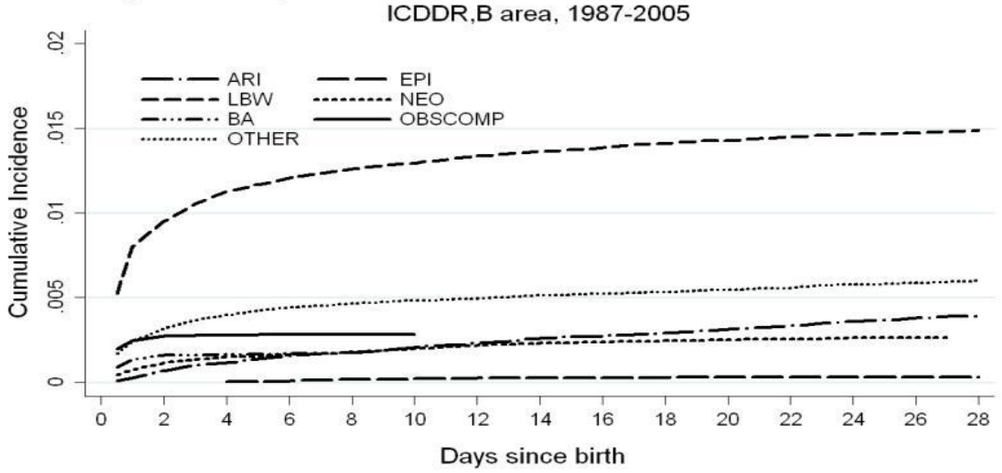


Figure 7: Nonparametric estimates of cause-specific neonatal death
comparison area, 1987-2005

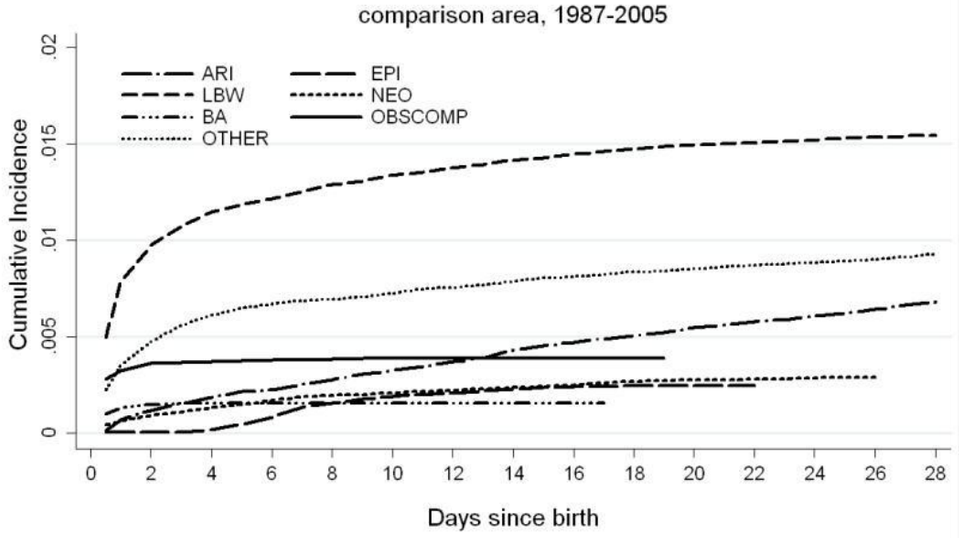


Figure 8:Parametric estimates of cause-specific neonatal mortality (ref. individuals), ICDDR,B area, 1987-1992

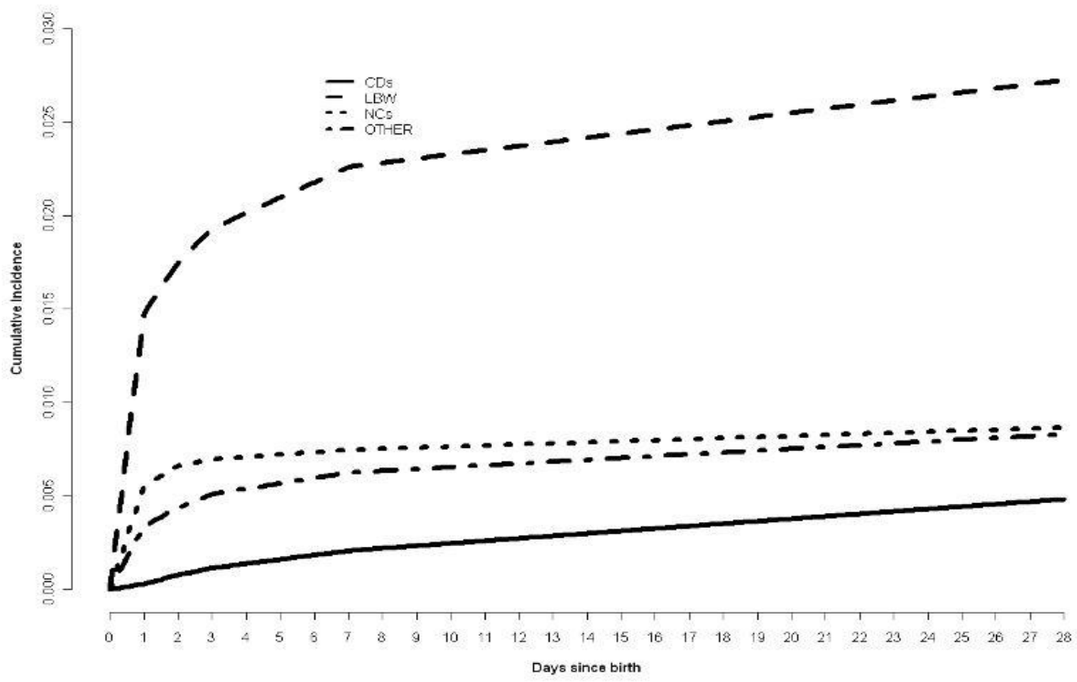


Figure 9:Parametric estimates of cause-specific neonatal mortality (ref. individuals), comparison area, 1987-1992

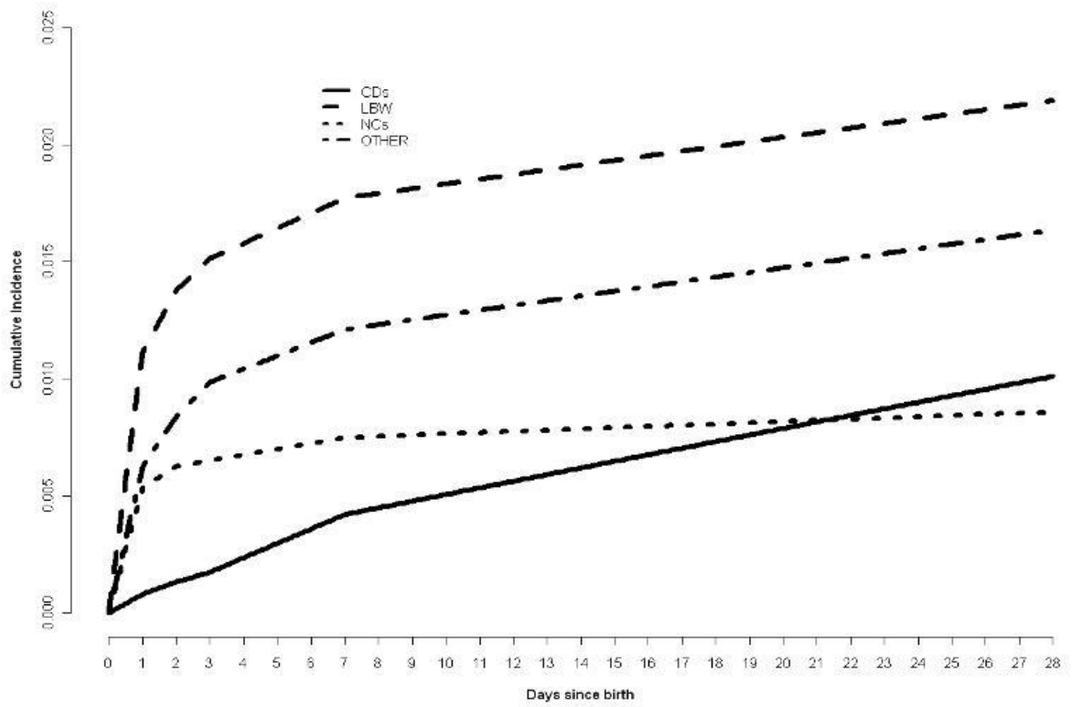
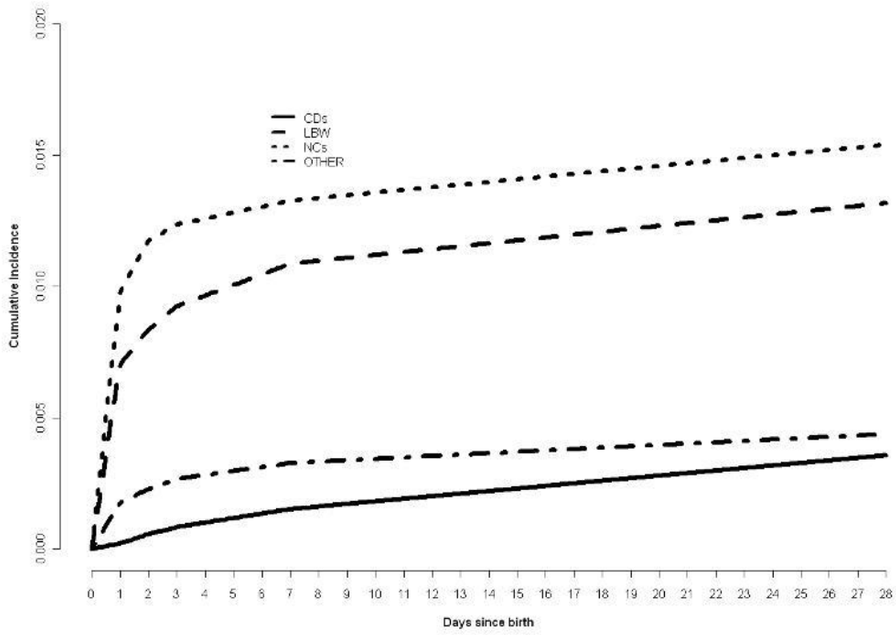


Figure 10:Parametric estimates of cause-specific neonatal mortality (ref. individuals), ICDDR,B area, 2000-2005



Annex

Table A1: Assignment of causes of neonatal death, 1987-2005, HDSS, Matlab, Bangladesh.

Codes (ICD9, ICD10)	Labels of code	Categories used (Table 2)	Categories used (Table 5 & 6)
190, 192, 193,452,458,P05,P07	Preterm delivery/low birth weight	Low birth weight (LBW)	LBW
454,P21	Birth asphyxia	Birth asphyxia (BA)	NCs
457, 456, P22-P29, P35,P36,P51,P76,P80	infections, respiratory and cardiovascular disorder specific to the perinatal period	Neonatal related conditions (NEO)	
449, Q01, Q02, Q03,Q24, Q35,Q37, Q42,Q45,Q89	Congenital abnormalities	Congenital abnormalities (CA)	
453, P15	Birth trauma	Birth trauma	
451,P00,P01,P02,P03	Obstetric complications	OBSCOMP	
P59	Haemorragic	Haemorragic	
P90-P96	Other disorders originated in the perinatal period	unspecified	OTHER
990,998,999,R34,R95,R96,R99	Unspecified causes		
R95,450,459	Sudden death		
K75, W75,X91, 293,344,420,460,461,552,555,559,691,738 ,	Other specific	Other specific	
010,013,014	Acute watery diarrhoea, dysentery, acute non-watery diarrhoea	Diarrhoea, dysentery	CDs
038, 046	Septicamia, viral hepatitis	Septicemia, hepatitis	
321, 325,328,191	Pneumonia, ALRI, Pneumonia with diarrhoea, Pneumonia severe	ARI	
A41,B01,G03,J11,J18, A03	Other bacterial diseases, viral infections characterized by skin and mucous membrane lesions	ARI	
456	EPI	EPI	

Chapter 6

Summary and conclusion

Child mortality in Bangladesh remains an important issue. Under-five mortality declined sharply during the last decades of the previous century, but the reduction is levelling off and child mortality is not declining fast enough to meet the Millennium Development Goal 4 of reducing under-five mortality by two-thirds between 1990 and 2015 (see United Nations 2001), so the further reduction of child mortality remains a significant challenge. Several hypotheses concerning possible causal mechanisms that increase or reduce child mortality were tested in the four papers included in the thesis.

The second chapter analysed infant mortality in Bangladesh, focusing on explaining death clustering within families, using prospective data from Matlab, Bangladesh, split into areas with and without extensive health services (the area covered by the International Centre for Diarrhoeal Disease Research: ICDDR,B and the comparison area, respectively). The modelling framework distinguished between two explanations of death clustering: (observed and unobserved) heterogeneity across families and a causal 'scarring' effect of the death of one infant on the survival chances of the next to be born. Keeping observed and unobserved characteristics constant, scarring was observed in the comparison area only. There the likelihood of infant death is about 29 per cent greater if the previous sibling died in infancy than otherwise. This effect mainly works through birth intervals: infant deaths are followed by shorter birth intervals, which increases the risk of infant death for the next child.

In the third chapter, using the same data sets as in first paper, we jointly analyzed infant mortality, birth spacing, and the probability of having another birth. To distinguish causal mechanisms from unobserved heterogeneity and reverse causality, this paper exploited dynamic panel data techniques, building on recent work by Bhalotra and van Soest (2008). The results are comparable between a treatment area with extensive health services and a comparison area with the standard health services provided by the government.

The results demonstrate that death at infancy of the previous child shortens the subsequent length of birth space by 49% in the ICDDR,B area and 46% in comparison area. As a result of replacement, every infant death in the comparison area leads to 0.54 births on average (and 0.51 births survive the first 12 months). In the ICDDR,B area, each infant death leads to 0.42 replacement births. The effects of the numbers of (surviving) boys and girls are consistent with son preference: having more surviving boys has a stronger positive effect on the birth interval than having more girls, though both effects are significant. Not having any surviving boys or girls leads to the shortest birth interval. Simulations on the basis of the estimated models

show how fertility and mortality can be reduced by, for example, breaking the causal link that leads to a short interval after a child has died.

In the fourth chapter, we investigated the causal role of contraceptive use on birth spacing and thereby on infant mortality. Using the same data set on the ICDDR,B area (since information on contraceptives is only available in that area), this paper extends the modelling framework used in the third chapter. The analysis is based on a three-part model: an equation explaining infant mortality, a model explaining whether contraceptives are used after a child is born (and if so, for how long), and an equation explaining birth intervals. Infant mortality is determined by covariates reflecting socio-economic status and other background characteristics, but also by the length of the preceding birth interval. Decisions about contraceptives are driven by similar covariates, but also by the survival status of the previous child and the family's gender composition. Birth spacing is driven by contraceptive use and other factors. Each part of the model incorporates unobserved mother specific heterogeneity. Results confirm the favourable effects of family planning programmes on child survival for second and higher birth orders that work through birth spacing. Our results imply that if family planning programmes would be expanded so that everyone would use contraceptives after each birth, a 7.9% reduction in infant mortality could be achieved – a reduction of 11 infant deaths per 1000 live births. This leads to the policy implication that strengthening family planning programs helps to reduce infant mortality.

The fifth chapter reports the underlying epidemiology of neonatal child deaths, taking into account the competing risks of neonatal deaths. A competing risk duration model is used incorporating both observed and unobserved mother specific heterogeneity, and assuming heterogeneity terms across various causes can be correlated. The results confirm the general conclusion on levels, trends and patterns of causes of neonatal deaths in the existing literature, but also reveal some remarkable socioeconomic differences in the risks of cause-specific deaths. Deaths due to low birth weight and other causes (sudden infant death, unspecified or specified) are better explained by the socio-economic covariates than deaths due to neonatal infections or obstetric complications.

The analysis in this thesis highlights the role of maternal and child health interventions (particularly tetanus toxoid immunization for pregnant women, nutrition programs, and high coverage health services / reduced distance to the nearest health centre). Policies that increase quality and equity in births may help to further reduce neonatal mortality.

Bibliography

Adjuik, M., Smith, T., Clark, S., Todd, J., Garrib, A., Kinfu, Y., Kahn, K., Mola, M., Ashraf, A., Masanja, H., Adazu, U., Sacarlal, J,, Alam, N., Maria, A., Gbangou, A., Mwageni, E., & Binka, F. (2006). Cause-specific mortality rates in sub-Saharan Africa and Bangladesh. *Bulletin of World Health Organization*, 84: 181-188.

Alam, N. (1995). Birth spacing and infant and early childhood mortality in a high fertility area of Bangladesh: Age–dependent and interactive effects. *Journal of Biosocial Science*, 27: 393-404.

Alam, N. & David, P.H. (1998). Infant and child mortality in Bangladesh: Age-specific effects of previous child's death. *Journal of Biosocial Science,* 30: 333-348.

Anwar, I., Killewo, J., Chowdhury, M.E., & Dasgupta, S.K. (2004). Bangladesh: Inequalities in utilization of maternal health care services – Evidence from Matlab. Discussion paper, Number 2, Dhaka: Health, Nutrition and Population.

Arulampalam, W. & Bhalotra, S. (2006). Sibling death clustering in India: State dependence versus unobserved heterogeneity. *Journal of the Royal Statistical Society A*, 169: 829-848.

Arulampalam, W. & Bhalotra, S. (2008). The linked survival prospects of siblings: Evidence from the Indian states. *Population Studies*, 62: 171-190.

Arulampalam, W. & Stewart, M.B. (2009). Simplified implementation of the heckman estimator of the dynamic probit model and a comparison with alternative estimators. *Oxford Bulletin of Economics and Statistics*, 71: 659-681.

Bairagi, R., Sutradhar, S.C., & Alam, N. (1999). Levels, trends and determinants of child mortality in Matlab, Bangladesh, 1966–1994. *Asia-Pacific Population Journal*, 14: 51–68.

Bairagi, R., Islam, M.M., & Barua, M.K. (2000). Contraceptive failure: Levels, trends and determinants in Matlab, Bangladesh. *Journal of Biosocial Science*, 32: 107-123.

Bang, A.T., Bang, R.A., Baitule, R.B., Reddy, H.M., & Deshmukh, M.D. (2005). Management of birth asphyxia in home deliveries in rural Gadchiroli: The effect of two types of birth attendants and of resuscitating with mouth-to-mouth, tube-mask or bag-mask. *Journal of Perinatology,* 25(Suppl): S82-S91.

Bari, W., Chowdhury, R.I., Islam, M.A., Chakraborty, N., & Akhter, H.A. (2002). The differentials and determinants of perinatal mortality in rural Bangladesh. *European Journal of Contraception and Reproductive Health Care,* 7: 216-222.

Baqui, A.H., Sabir. A.A., Begum. N., Arifeen. S.E., Mitra. S.N., & Black, R.E. (2001). Causes of childhood deaths in Bangladesh: An update. *Acta Paediatrica*, 90: 682-690.

Ben-Porath, Y. (1976). Fertility response to child mortality: Micro data from Israel. *The Journal of Political Economy*, l84: S163-S178.

Bhalotra, S. & van Soest, A. (2008). Birth spacing, fertility and neonatal mortality in India: Dynamics, frailty and fecundity. *Journal of Econometrics*, 143: 274-290.

Bhalotra, S., Valente, C., & van Soest, A. (2010a). Religion and childhood death in India. In: Basant, R. and Sharif, A. (eds.). *Handbook of Muslims in India*. Delhi: Oxford University Press: 123-165.

Bhalotra, S., Valente, C., & van Soest, A. (2010b). The puzzle of Muslim advantage in child survival in India. *Journal of Health Economics,* 29: 191-204.

Bhatia, S. (1981). Traditional childbirth practices: Implications for a rural MCH program. *Studies in Family Planning*, 12: 66-75.

Bhatia, S. (1983). Contraceptive users in rural Bangladesh - a time trend analysis. *Studies in Family Planning*, 14: 20-28.

Bhatia, S. (1989). Patterns and causes of neonatal and postneonatal mortality in rural Bangladesh. *Studies in Family Planning*, 20: 136-146.

Bhuiya, A., Zimicki, S., & D`Souza, S. (1986). Socioeconomic differentials in child nutrition and morbidity in a rural area of Bangladesh. *Journal of Tropical Pediatrics*, 32: 17-23.

Blinder, A. S. (1973). Wage discrimination: Reduced form and structural estimates. *Journal of Human Resources*, 8: 436–455.

Bolstad, W. M. & Manda, S.O. (2001). Investigating child mortality in Malawi using family and community random effects: A bayesian analysis. *Journal of the American Statistical Association*, 96: 12-19.

Bongaarts, J. (1987). Does family planning reduce infant mortality? Comment. *Population and Development Review*, 13: 323-334.

Bryce, J., Cynthia, B.P., Shibuya, K., & Black, R.E. (2005). WHO child health epidemiology reference group, WHO estimates of the causes of death in children. *Lancet*, 365: 1147-1152.

Butler, J. S. & Moffitt, R. (1982). A computationally efficient quadrature procedure for the one-factor multinomial probit model. *Econometrica*, 50: 761-764.

Caldwell, J.C. (1993). Health transition: The cultural, social and behavioural determinants of health in the Third World. *Social Science & Medicine*, 36: 125-135.

Caldwell, J.C. & Caldwell, P. (1978). The achieved small family: Early fertility transition in an African city. *Studies in Family Planning*, 9: 2-18.

Caldwell, J.C., Findley, S., Caldwell, P., Santow, G., Cosford, W., Braid, J., & Broers-Freeman, D. (eds). (1990). What we know about health transition: The cultural, social and behavioural determinants of health. Volumes 2, Canberra: Australian National University.

Chen, L.C., Rahman, M., & Sarder, A.M. (1980). Epidemiology and causes of death among children in a rural area of Bangladesh. *International Journal of Epidemiology*, 9: 25-33.

Chowdhury, A.K.M., Khan, A.R. & Chen, L. (1976). The effect of child mortality experience on subsequent fertility: In Pakistan and Bangladesh. *Population Studies*, 30: 263-278.

Chowdhury, H.R., Thompson, S., Ali, M., Alam, N., Yunus, M., & Streatfield, P.K. (2010). Causes of neonatal deaths in a rural subdistrict of Bangladesh: Implications for intervention. *Journal of Health Population Nutrition*, 28: 375-382.

Chowdhury, M.K. & Bairagi, R. (1990). Son preference and fertility in Bangladesh. *Population and Development Review*, 16: 749-757.

Cleland, J. & Sathar, Z.A. (1984). The effect of birth spacing on childhood mortality in Pakistan. *Population Studies*, 38: 401-418.

Conde-Agudelo, A.,Belizan, J.M., & Diaz-Rossello, J. (2003). Kangaroo mother care to reduce morbidity and mortality in low birth weight infants (Cochrane Review). In: *The Cochrane Library*. Issue 4, Oxford: Update Software.

Conde-Agudelo, A., Rosas-Bermudez, A., & Kafury-Goeta, A.C. (2006). Birth spacing and risk of adverse perinatal outcomes: a meta-analysis. *Journal of the American Medical Association*, 295: 1809-1823.

Cornfield, J. (1957). The estimation of the probability of developing a disease in the presence of competing risks. *Disease Probability,* 47: 601-607.

Coviello, V. & Boggess, M. (2004). Cumulative incidence estimation in the presence of competing risks. *The Stata Journal,* 4: 103-112.

Cox, D.R. (1959). The analysis of exponentially distributed life-times with two types of failures. *Journal of the Royal Statistical Society* B, 21: 411-421.

Curtis, S.L., Diamond, I. & McDonald, J.W. (1993). Birth interval and family effects on postneonatal mortality in Brazil. *Demography,* 30: 33-43.

DasGupta, M. (1990). Death clustering, mothers' education and the determinants of child mortality in rural Punjab. *India Population Studies,* 44: 489-505.

Datta, A.K., & Bairagi, R. (2000). Improvement in female survival: A quite revolution in Bangladesh. *Asia Pacific Population Journal,* 15: 19-40.

DaVanzo, J. & Starbird, E.H. (1991). Correlates of short interbirth intervals in Peninsular Malaysia: Their pathways of influence through breastfeeding and contraceptive use. *Studies in Family Planning,* 22: 241-254.

DaVanzo, J. & Pebley, A.R. (1993). Maternal depletion and child survival in Guatemala and Malayasia. Santa Monica: Rand Corporation.

DaVanzo, J., Razzaque, A., Rahman, M., & Hale, L. (2004). The effects of birth spacing on infant and child mortality, pregnancy outcomes, and maternal morbidity and mortality in Matlab, Bangladesh. Santa Monica: Rand Corporation.

DaVanzo, J., Hale, L., Razzaque, A., & Rahman, M. (2007). Effects of interpregnancy interval and outcome of the preceding pregnancy on pregnancy outcomes in Matlab, Bangladesh. *British Journal of Obstetrics and Gynaecology,* 114: 1079-1087.

DaVanzo, J., Hale, L., Razzaque, A., & Rahman, M. (2008). The effects of pregnancy spacing on infant and child mortality in Matlab, Bangladesh: How they vary by the type of pregnancy outcome that began the interval. *Population Studies*, 62: 131-154.

D'Souza, S. (1981). A population laboratory for studying disease processes and mortality- the demographic surveillance system, Matlab Comilla, Bangladesh. *Rural Demography,* 8: 29-51.

D'Souza, S. and A. Bhuiya. (1982). Socioeconomic mortality differentials in a rural area of Bangladesh. *Population and Development Review*, 8: 753-769.

Fairlie, R.W. (2005). An Extension of the blinder-oaxaca decomposition technique to logit and probit models. *Journal of Economic and Social Measurement,* 30: 305-316.

Fauveau, V., Koenig, M.A., & Wojtyniak, B. (1991). Excess female deaths among rural Bangladeshi children: An examination of cause-specific mortality and morbidity. *International Journal of Epidemiology,* 20: 729-735.

Fauveau, V., Wojtyniak, B., Chowdhury, H.R.,& Sarder, A.M. (1994). Assessment of cause of death in the Matlab Demographic Surveillance System. In: Fauveau V (eds.). *Matlab: Women, Children and Health.* Dhaka: International Centre for Diarrhoeal Diseases Research: 65-86.

Fine, J.P. & Gray, R.J. (1999). A proportional hazards model for the subdistribution of a competing risk. *Journal of the American Statistical Association,* 94: 496-509.

Fotouhi, A. R. (2005). The initial condition problem in longitudinal binary process: A simulation study. *Simulation Modelling Practice and Theory,* 13: 566-583.

Goldberg, I.D., Lovin, M.L., Gerhardt, P.R., Handy, V.H., & Cashman, R.E. (1956). The probability of developing cancer. *Journal of the National Cancer Institute,* 17: 155-173.

Gubhaju, B. B. (1985). The effect of previous child death on infant and child mortality in rural Nepal. *Studies in Family Planning,* 16: 231-236.

Guo, G. (1993). Use of sibling data to estimate family mortality effects in Guatemala. *Demography,* 30: 15-32.

Guo, G. & Rodriguez, G. (1992). Estimating a multivariate proportional hazard model via the EM algorithm, with an application to child survival in Guatemala. *Journal of the American Statistical Association,* 87: 969-976.

Haaga, J. G. (1991). Mechanisms for the association of maternal age, parity, and birth spacing with infant health. Santa Monica: RAND Corporation.

Hale, L., DaVanzo, J., Razzaque, A., & Rahman, M. (2006). Why are infant and child mortality rates lower in the MCH-FP area of Matlab, Bangladesh? *Studies in Family Planning*, 37: 281-292.

Hale, L., DaVanzo, J., & Razzaque, A. (2009). Which factors explain the decline in infant and child mortality in Matlab, Bangladesh? *Journal of Population Research,* 26: 3-20.

Hajivassiliou, V. A., & Ruud, P.A.(1994). Classical estimation methods for LDV models using simulation. In: Robert F. Engle and Daniel L. McFadden (eds.). *Handbook of Econometrics.* Volume IV, Amsterdam: North-Holland: 2384-2438.

Harrington, J.A. (1971). The effect of high infant and childhood mortality on fertility: The west African case. *Concerned Demography*, 3: 22-35.

Heckman, J.J. (1981). The incidental parameters problem and the problem of initial conditions in estimating a discrete time – discrete data stochastic process. In: C.F. Manski and D., McFadden (eds.). *Structural Analysis of discrete data with econometric applications*. Cambridge: MIT Press: 114-178.

Heckman, J.J., Holtz, J., & Walker, J.R. (1985). New evidence on the timing and spacing of births. *American Economic Review,* 75: 179-184.

Hobcraft, J. N., McDonald, J.W., & Rutstein, S.O. (1983). Childspacing effects on infant and early child mortality. *Population Index,* 49: 585-618.

Hobcraft, J. N., McDonald, J.W., & Rutstein, S.O. (1985). Demographic determinants of infant and early child mortality: A comparative analysis. *Population Studies,* 39: 363-385.

Huffman, S. L., Kathleen, F., Hubert A. A., & Streble, P. (1987). Nutrition and fertility in Bangladesh: Breastfeeding and post partum amenorrhoea. *Population Studies,* 41: 447-462.

ICDDR,B Centre for Health and Population Research, Dhaka, Bangladesh. (2006). Health and Demographic Surveillance System-Matlab: Registration of health and demographic events 2004. Scientific Report 93.

Jones, G, Steketee, R., Black, R., Bhutta, Z., & Morris, S. (2003). How many child deaths can we prevent this year? *Lancet,* 362: 65-71.

Karar, Z.A., Alam, N., & Streatfield, P.K. (2009). Epidemiological transition in rural Bangladesh, 1986-20*06. Global Health Action,* DOI:10.3402/gha.v2i0.1904.

Knodel, J. (1978). European populations in the past: Family-level relations. In: S.H. Preston (eds.). *The effects of infant and child mortality on fertility.* New York: Population Division, United Nations.

Koenig, M.A., Phillips, J.F., Campbell, O.M., & D'Souza, S. (1990). Birth intervals and childhood mortality in rural Bangladesh. *Demography*, 27: 251-265.

Koenig, M. A., Rob, U., Khan, M.A., Chakraborty, J., & Fauveau, V. (1992). Contraceptive use in Matlab, Bangladesh in 1990: Levels, trends, and expectations. *Studies in Family Planning*, 23: 352-364.

Lancaster, T. (1979). Econometric methods for the duration of unemployment. *Econometrica*, 47: 939-956.

Lawn, J.E., Cousens, S., & Zupan, J. (2005). 4 million neonatal deaths: When? Where? Why? *Lancet*, 365: 891-900.

Lawn, J.E., Wilczynska, K., & Cousens, S.N. (2006). Estimating the causes of 4 million neonatal deaths in the year 2000. *International Journal of Epidemiology*, 35: 706-718.

LeGrand, T.K. & Phillips, J.F. (1996). The effect of fertility reductions on infant and child mortality: Evidence from Matlab in rural Bangladesh. *Population Studies*, 50: 51-68.

Madise, N.J. & Diamond, I. (1995). Determinants of infant mortality in Malawi: An analysis to control for death clustering within families. *Journal of Biosocial Science*, 27: 95-106.

Majumder, A. K., May, M., & Pant, P.D. (1997). Infant and child mortality determinants in Bangladesh: Are they changing? *Journal of Biosocial Science*, 29: 385-399.

Manton, K.G., Stallard, E., & Vaupel, J. W. (1981). Methods for the mortality experience of heterogeous populations. *Demography*, 18: 389-410.

Matthiesson, P.C., & McCann, J.C. (1976). The role of mortality in the European fertility transition: Aggregate-level relations. In: S.H. Preston (ed.). *The effects of infant and child mortality on fertility*. New York: Academic Press: 47-68.

Miller, J.E., Trussell, J., Pebley, R.A., & Vaughan, B. (1992). Birth spacing and child mortality in Bangladesh and Philippines. *Demography*, 29: 305-318.

Moffitt, R. (2005). Remarks on the analysis of causal relationships in population research. *Demography*, 42: 91-108.

Mondot-Bernard, J.M. (1977). Relationships between fertility, child Mortality and nutrition in Africa. Tentative analysis. Technical papers, Paris: Development Centre of OECD.

Mosley, W.H. & Chen, L.C. (1984). An analytical framework for the study of child survival in developing countries. *Population Development Review*, 10(Suppl.): 25-45.

National Institute of Population Research and Training (NIPORT), Mitra and Associates, and ORC Macro. (2005). Bangladesh Demographic Health Survey 2004. Dhaka, Bangladesh and Calverton, Maryland [USA].

National Institute of Population Research and Training (NIPORT), Mitra and Associates, and ORC Macro. (2008). Bangladesh Demographic Health Survey 2007. Dhaka, Bangladesh and Calverton, Maryland [USA].

Nyarko, P., Madise, N.J., & Diamond, I. (2003). Child loss and fertility behaviour in Ghana. Southampton, UK: Social Statistics Research Centre, S3RI Application and Policy Working Paper, A03/08, p. 25.

Oaxaca, R. (1973). Male-female wage differentials in urban labor markets. *International Economic Review*, 14: 693–709.

Omariba, D. W. R., Fernando R., & Roderic, B. (2008). Correlated mortality of siblings in Kenya: The role of state dependence. *Demographic Research*, 18: 311-336.

Omran, A.R. (1982). Epidemiological transition: Theory. In: Ross J.A. (eds.). *International Encyclopedia of Population.* Volume 1, New York: The Free Press: 172-75.

Park, C.B., Islam, M.A., Chakraborty, N., & Kantner, A. (1998). Partitioning the effect of infant and child death on subsequent fertility: An exploration in Bangladesh. *Population Studies*, 52: 345-356.

Phillips, J.F., Simmons, R., Chakraborty, J., & Chowdhury, A.I. (1984). Integrating health services into an MCH-FP program: Lessons from Matlab, Bangladesh. *Studies in Family Planning*, 15: 153-161.

Phillips, J.F., Simons, R., Koenig, M.A., & Chakraborty, J. (1988). Determinants of reproductive change in a traditional society: Evidence from Matlab, Bangladesh. *Studies in Family Planning*, 19: 313-334.

Phillips, J. F., Wayne, S. S., Shushum, B., Rahman, M., & Chakraborty, J. (1982). The demographic impact of the family planning-health services project in Matlab, Bangladesh. *Studies in Family Planning*, 13: 131-140.

Potter, J. E. (1988). Does family planning reduce infant mortality? Comment. *Population and Development Review*, 14: 179-187.

Pritchett, H.L. (1994). Desired fertility and the impact of population policies. *Population and Development Review*, 20: 1-55.

Rahman, M., Akbar, J., Phillips, J.F., & Becker, S. (1992). Contraceptive use in Matlab, Bangladesh: The role of gender preference. *Studies in Family Planning*, 23: 229-242.

Rahman, M. & DaVanzo J. (1993). Gender preference and birth spacing in Matlab, Bangladesh. *Demography*, 30: 315-332.

Rahman, A., Iqbal, Z., Bunn, J., Lovel, H., & Harrington, R. (2004). Impact of maternal depression on infant nutritional status and illness: A cohort study. *Archives of General Psychiatry*, 61: 946-952.

Razzaque, A., Streatfield, P.K., & Gwatkin, D.R. (2007). Does health intervention improve socioeconomic inequalities of neonatal, infant and child mortality? Evidence from Matlab, Bangladesh. *International Journal for Equity in Health*, 6: 1-7.

Reddiah,V.P. & Kapoor, S.K. (1988). Acute respiratoty infection in rural under-fives. *Indian Journal of Pediatrics*, 55: 424-426.

Retherford, R.D., Choe, M.K., Shyam, T., & Gubhaju, B.B. (1989). To what extent does breastfeeding explain birth-interval effects on early childhood mortality? *Demography*, 26: 439-450.

Ronsmans, C., Chowdhury, M.E., DasGupta, S.K., Ahmed, A., & Koblinsky, M. (2010). Effect of parent's death on child survival in rural Bangladesh: A cohort study. *Lancet*, 375: 2024-2030.

Rosenblum, D. (2009). Economic causes of sex discrimination in India, PhD thesis. Yale University.

Rosenzweig, M.R. & Wolpin, K.I. (1988). Heterogeneity, intrafamily distribution, and child health. *Journal of Human Resources*, 23: 437-461.

Rutstein, S. (2005). Effects of preceding birth and pregnancy intervals on child mortality and nutritional status in less developed countries: Evidence from the demographic and health surveys. *International Journal of Gynecology and Obstetrics*, 89: S7-S24.

Saha, U.R., Kamal, N., & Bairagi, R. (2004). Discontinuation of contraceptives and method switching in rural Bangladesh. *Sharing Population and Development Research across South and West Asia*. Fifth Annual Research Conference Proceedings, 14-16 December, 2004.

Saha, U.R. & van Soest A. (2011). Infant death clustering in families: Magnitude, causes, and the influence of better health services, Bangladesh 1982-2005. *Population Studies*, 65: 273-287.

Santow, G., & Bracher M. D. (1984). Child death and time to the next birth in Central Java. *Population Studies*, 38: 241-53.

Sastry, N. (1997). Family-level clustering of childhood mortality risk in Northeast Brazil. *Population Studies,* 51: 245-261.

Scrimshaw, N. (1996). Nutrition and health from womb to tomb. *Nutrition Today*, 31: 55-67.

Smith, D. P. (1985). Breastfeeding, contraception, and birth intervals in developing countries. *Studies in Family Planning*, 16: 154-163.

Stange, K. (2011). A longitudinal analysis of the relationship between fertility timing and schooling. *Demography,* 48: 931-956.

Steele, F. & Diamond, I. (1999). Contraceptive switching in Bangladesh. *Studies in Family Planning*, 30: 315-328.

Steer, R. A., Scholl, T.O., Hediger, M.L., Fischer, R.L. (1992). Self-reported depression and negative pregnancy outcomes. *Journal of Clinical Epidemiology*, 45: 1093-1099.

Stewart, M. B. 2007. The interrelated dynamics of unemployment and low-wage employment. *Journal of Applied Econometrics*, 22: 511-531.

Sufian, A. J. M., & Johnson, N.E. (1989). Son preference and child replacement in Bangladesh: A new look at the child survival hypothesis. *Journal of Biosocial Science*, 21: 207-16.

Swenson, I. (1978). Early childhood survivorship related to the subsequent interpregnancy interval and outcome of the subsequent pregnancy. *Journal of Tropical Pediatrics,* 24: 103-106.

Train, K.E. (2003). Discrete choice methods with simulation. Cambridge: Cambridge University Press.

Trussell, J. (1988). Does family planning reduce infant mortality? Comment. *Population and Development review*, 14: 171-178.

UNDP (2003). Millennium development goals: A compact among nations to end human poverty. Human development report 2003.

United Nations (2001). General Assembly, 56th session. Road map towards the implementation of the United Nations millennium declaration: Report of the Secretary-General. New York: United Nations (UN document no. A/56/326).

Van Ginneken, J. K. (1974). Breastfeeding as a birth spacing method. *Studies in Family Planning*, 5: 201-206.

Van Ginneken, J.K., Bairagi, R., Francisco, A. De, Sarder, A.M., & Vaughan, P. (1998). Health and demographic surveillance in Matlab: Past, present, and future. Scientific Publication Number 72.

Velaphi, S. & Pattinson, R. (2007). Avoidable factors and causes of neonatal deaths from perinatal asphyxia-hypoxia in South Africa: National perinatal survey. *Annals of Tropical Paediatrics: International Child Health*, 27: 99-106.

Victora, C. G., Barros, F. C., & Vaughan. J. P. (2001). The impact of health interventions on inequalities: Infant and child health in Brazil. In: D. A. Leon and G. Walt (eds.). *Poverty, Inequality and Health: An International Perspective*. Oxford: Oxford University Press: 125-136.

Waldron, I. (1983). Sex differences in human mortality. *Social Science & Medicine,* 17: 321-33.

Whitworth, A. & Stepheson, R. (2002). Birth spacing, sibling rivalry and child mortality in India. *Social Science & Medicine,* 55: 2107-2119.

Wolpin, K.I. (1978). An economic analysis of crime and punishment in England and Wales. *Journal of Political Economy*, 86: 815-840.

Wolpin, K. I. (1997). Determinants and consequences of the mortality and health of infants and children. In: M. Rosenzweig and O. Stark, (eds.). *Handbook of Population and Family Economics.* Vol. 1A, Amsterdam: North Holland: 483-557.

Wolpin, K.I. (1998). The impact of infant and child mortality risk on fertility. In: Committee on Population, National Research Council (eds.). *From Death to Birth*. Washington, D.C.: National Academy Press: 74-111.

World Health Organization. (1977). Manual of the international statistical classification of diseases, injuries, and causes of death. 9th revision. Geneva*:* World Health Organization.

Yeakey, M.P., Muntifering, C.J., Ramachandran, D.V., Myint, Y., Creanga, A.A. & Tsui. A.O. (2009). How contraceptive use affects birth intervals: Results of a literature review. *Studies in Family Planning*, 40: 205-214.

Yun, M.S. (2004). Decomposing differences in the first moment. *Economics Letters*, 82: 275-280.

Zaba, B. & David, P.H. (1996). Fertility and the distribution of child mortality risk among women: An illustrative analysis. *Population Studies*, 50: 263–278.

Zenger, E. (1993). Siblings' neonatal mortality risks and birth spacing in Bangladesh. *Demography*, 30: 477-488.

Zimmer, B.G. (1979). Consequences of the number and spacing of pregnancies on outcome. and of pregnancy outcome on spacing. *Social Biology*, 26: 161-78.